DISEASES OF THE EAR

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PREFACE TO THE THIRD EDITION.

THE purpose of successive revisions of a medical text book is naturally to make it as nearly representative, as the writer's ability permits, of whatever is significant in recent thought or research.

While the comparatively rapid exhaustion of the second edition has made any re-writing of chapters—had that been advisable—out of the question, a conscientious effort has been made to interpolate whatever of recent work has been of approved interest or value. The new matter has been carefully indexed, and it is hoped that these additions, with the bibliography relating thereto, may give to this edition a greater value as a practical book of reference.

The relatively recent studies and writing of Dr. A. A. Gray of Glasgow on the nature and pathology of oto-sclerosis and the widespread revival of Anglo-Saxon interest resulting therefrom, the developing theories as to the physiology of the so-called "otolith mechanism," now under investigation by the Dutch School of Otologists, the renewed interest of our British collegues in the conflicting theories of tone perception, and the obvious tendency toward a broader neurologic investigation of intracranial lesions of otitic origin give to the student and practitioner of otology much food for stimulating thought and speculation.

One new chapter—on TUMORS OF THE 8TH NERVE—represents an extension of the field usually covered by text books on otology.

With the constantly broadening concept of medical science as a whole, the merging or overlapping of related branches has been one of the most conspicuous tendencies of recent years. As neurology has borrowed and assimilated the results of Barany's work on the physiology of the labyrinth, so of necessity has otology been forced at certain points to invade the domain of neurology.

In a relatively recent paper, a distinguished surgeon, speaking of tumors of the acoustic nerve, brought otologists under the implied reproach of having usually failed with respect to this lesion even "to recognize the presence of a tumor at all." Clearly, this statement, or its implications, if justified, would bring discredit upon otology. If we accept the fact that the eighth nerve, quite as much as the drum membrane or ossicles is part and parcel of the organ or mechanism

of hearing, it is clear that a working knowledge of this very grave lesion should not be confined to special students of oto-neurology, but should belong quite generally to the rank and file of otologists. Lack of such knowledge may lead an otherwise competent aurist into serious errors of diagnosis and treatment.

The author believes that no addition to this book could be of greater interest or value to practical otologists than a chapter giving in condensed form the accepted facts as to the incidence, gross pathology, prognosis, and particularly as to the symptomatology, of *tumors of the eighth nerve.*

THE AUTHOR

JUNE, 1923.

PREFACE TO SECOND EDITION

WHILE the past few years have been marked by no startling developments in otology, advances of a practical order have not been wholly wanting. Our conception of the pathology, processes of repair and rationale of treatment of certain lesions has undergone very considerable modification. The mastoid operation of a few years ago was a bone operation pure and simple. To-day the plastic work vies in practical importance with that upon the temporal bone itself. In other words, our conception of the important rôle which the soft parts may play in post-operative tissue repair has led logically to a different surgical technic, which, in turn, has given us better and quicker results. To record these developments parts of this chapter have been practically rewritten. Again, the further study of the changes in the cerebrospinal fluid occurring in various diseases has conduced materially to a more correct interpretation of the meningeal lesions and disorders of otitic origin and has brought to light certain sources of error in diagnosis. Beyond such changes and additions as were necessary to bring these practical phases of otology in line with modern ideas, alterations in the text and arrangement of the subject matter have been avoided.

Two new chapters have been added, of which the first is devoted to Barany's theory of the cerebellar control of joint movements, with a discussion of the pointing tests in their relation to vestibular and cerebellar disease. The second includes a brief account of the various types of war deafness,—deafness due to direct injury of the skull, nerve deafness due to loud explosive noises, concussion deafness due to the air displacements of near-by explosions, psychic disturbances of function, pithiatism, etc.,—phenomena which constitute for most of us a practically new chapter in aural disease. Direct products of military hardship and injuries, as these lesions and functional disorders undoubtedly are, their relation to certain phases of some of the commoner ear lesions is sure to provide a fruitful field for future investigation and discussion. It is hoped, therefore, that the changes and additions which distinguish this edition from the first may prove to be of distinct practical usefulness to students and practitioners of otology

PHILIP D. KERRISON.

PREFACE

Probably in no branch of medicine have more notable advances been achieved during the past decade than in otology. The wholly new field of work which has been opened to us by the successful investigation of the static labyrinth; the new light upon syphilitic lesions of the labyrinth and auditory nerve resulting from the recent world-wide renewal of interest in the study of all phases of syphilis; and the investigations still in progress as to the influence of autogenous vaccines and leucocyte extracts upon certain phases of aural disease,—these and other additions to our knowledge have suggested new problems in the working out of which laboratory investigations have been closely followed by practical therapeutic results.

In aural surgery our activities can no longer be confined to the narrow limits of the tympanum and mastoid process, but must include the more hazardous field of intracranial surgery and the yet more delicate and difficult work upon the auditory labyrinth itself. There is, then, some justification at the present time for yet another book,—*i.e.*, an attempt to present the complex subject of otology in the light of recent advances.

If the arrangement of the subject matter, particularly as to the proportionate space given to the different subjects, represents a departure from that usually followed, this has become necessary in order to bring the various subjects now pertaining to otology into a true relation with their actual and proportionate importance.

For example, labyrinthine physiology, suppurative diseases of the labyrinth, and the surgery of the labyrinth, which are usually rather briefly dealt with, are here considered in three separate chapters which occupy a very considerable section of the book. Again, the suppurative lesions of the brain and meninges are accorded far more space than is usually allotted them in text-books of otology.

In the Section devoted to operative surgery, the plan of illustrating each successive step of the various operations has been adopted. This, it is believed, will be of special value to students of otology living at a distance from the larger medical centres and who are therefore denied the educational advantages of large surgical clinics. With few exceptions, the illustrations throughout the book are from original drawings made under the writer's personal supervision.

PREFACE

In the preparation of this manual the writer has endeavored to give greatest prominence to the practical side of otology. This has necessitated the elimination of many time-honored but discarded theories and therapeutic measures which no longer have any bearing upon the practice of otology.

In conclusion, I wish to express my thanks to Dr. J. H. Guntzer for many valuable abstracts from the literature, and to Miss Eleanor Fry, whose clever and anatomically correct drawings form so attractive and valuable a feature of this book. I wish also to express my indebtedness to the publishers, Messrs. J. B. Lippincott Company, for many helpful suggestions as to the arrangement of the text.

Philip D. Kerrison.

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DISEASES OF THE EAR

CHAPTER I.

ANATOMY AND PHYSIOLOGY OF THE SOUND-CONDUCTING APPARATUS.

Anatomically, the ear may be divided into three parts,—viz., the outer ear (auricle and external auditory meatus), the middle ear (tympanum and structures contained therein), and the inner ear, or labyrinth. Physiologically, the organ of hearing must be regarded as consisting of two distinct mechanisms,—viz. (1) a sound-conducting apparatus, having to do solely with the conduction, or transmission, of sound waves; and (2) a perceptive mechanism, an organ specialized for the reception and analysis of sound waves, and their appreciation as sound.

From the view-point of the otologist, the latter is much the more practical and useful division, suggesting at once the necessary separation of all aural disorders into two main groups,—viz. (1) those which originate in, or involve chiefly, the labyrinth or end-organs of the auditory nerve (labyrinthine diseases); and (2) those depending upon pathological changes in some portion of the conducting mechanism (diseases of the conducting apparatus).

The *conducting apparatus* is composed of the following structures: the auricle and external auditory meatus; the drum membrane, and ossicular chain with the muscles and ligaments attached thereto; the tympanic cavity in which the ossicles are suspended, and the Eustachian tube.

The *perceptive apparatus* includes: the auditory nerve, its nuclei of origin, trunk, association fibres, cortical nuclei, its terminal fibres in the cochlea, and the membranous cochlea itself.

The Auricle, or Pinna (Fig. 1).—The auricle is an irregularly ovoid structure which might well be described as an outer terminal expansion of the cartilaginous meatus. It is roughly convex posteriorly, concave anteriorly. It consists chiefly of a thin cartilaginous plate, covered by perichondrium, to which the integument is closely adherent. The lower dependent portion, containing no cartilage, is composed of connective tissue containing some fat cells, and is called the *lobule*, or lobe of the ear (Fig. 1, 10). The free margin of the cartilaginous plate is curled toward the anterior surface of the auricle, forming a narrow ridge which is called the *helix* (1). The helix begins in front just above the orifice of the meatus, and ends behind at a point which marks the beginning of the posterior border of the lobule. Separated from the helix by a narrow curvilinear depression (the *fossa of the helix*, 2), and parallel with its posterior portion, is another elevation, known as the *antihelix* (4). The antihelix divides above into two arms, or crura, which inclose a triangular depression, the

fossa of the antihelix (3). The antihelix terminates below in a prominence directed forward and upward, called the *antitragus* (8). Opposite this, but directed backward and somewhat overlapping the orifice of the meatus, is another projecting lamella of cartilage, the *tragus* (7). The tragus and antitragus are separated below by a deep notch, the *incisura intertragica* (9). These structures—viz., the antihelix above and behind, the antitragus and incisura intertragica below, and the tragus in front—form the boundaries of a deep central depression, the *concha* (5). In the anterior part of the floor of this concavity is the orifice of the *external auditory canal* (6).

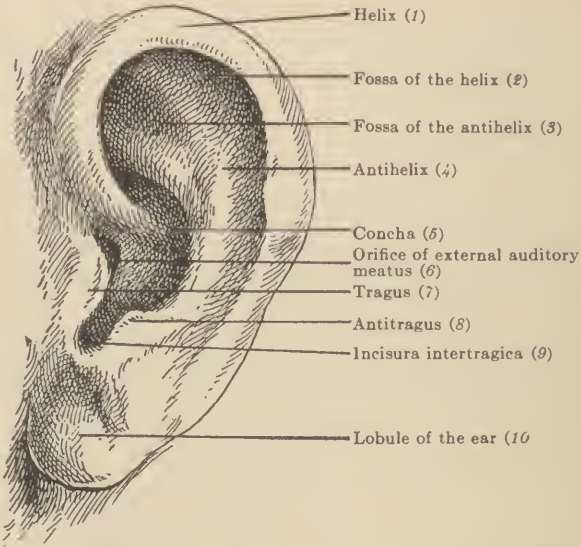


Fig. 1.—The auricle.

The auricle varies greatly in size and shape, and also in the angle which its posterior surface forms with the side of the skull. Its cartilaginous frame consists of a single plate of reticulated cartilage, the various folds of which assist in the formation of the elevations and depressions described above (Fig. 2).

Muscles of the Auricle.—These consist of two sets,—viz. (A) those which arise from the side of the head and are inserted into some portion of the auricle, their contraction serving to move the auricle as a whole; and (B) those which have both origin and insertion upon the auricle itself, their contraction serving to change its form or shape.

(A) The muscles arising from the side of the head are three in number, —viz., the *attrahens aurem*, or auricularis anterior; the *attolens aurem*, or auricularis superior, and the *retrahens aurem*, or auricularis posterior. While man in the process of evolution has lost the voluntary control of these muscles, there are some partial exceptions to this rule, certain individuals being able to move the ear in one or more directions (see Fig. 3).

(B) The second group of muscles—those having no attachment other than to the auricle itself—are six in number. Four of these—the helicis major, helicis minor, tragicus, and antitragicus—are attached to the anterior, concave surface of the auricle, the other two—the transversus

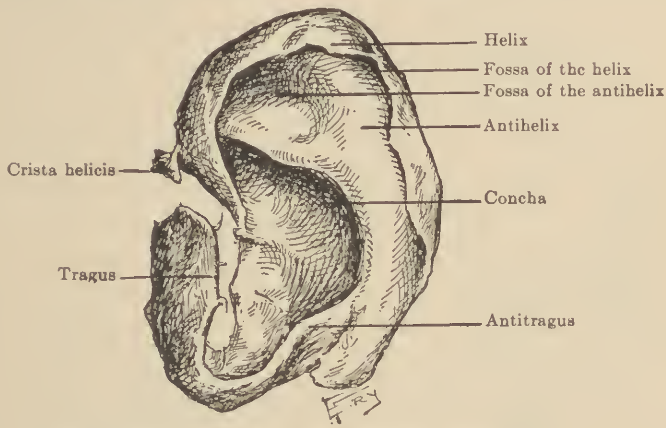


Fig. 2.—Auricular cartilage (from dissection by the author).

auriculæ and obliquus auris—being confined to its posterior surface. With the exception of the transversus auriculæ and the obliquus auris, the form and distribution of all these muscles are clearly shown in the accompanying illustration (Fig. 3).

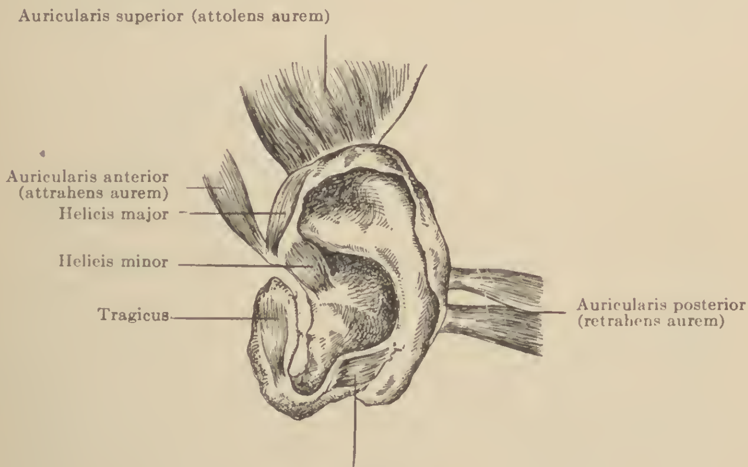


Fig. 3.—The auricular muscles.

The External Auditory Meatus.—The external canal in the adult consists of two parts,—(1) a membrano-cartilaginous tube, and (2) an inner bony tube, to which the former is joined.

The membrano-cartilaginous canal (Fig. 4) is an inward tubular extension of the auricle. Its external orifice is just behind the tragus in the anterior part of the floor of the concha. Its cartilaginous walls are deficient in the upper and posterior part of the canal, this deficiency being supplied by firm connective tissue which is continuous internally with the periosteal lining of the bony meatus. The cartilaginous frame of the meatus consists of an irregularly tongue-shaped plate of cartilage, curved longitudinally into a tortuous trough, which gives form to the outer half of the external canal. Its outer side, or base, which is continuous with the cartilaginous concha, is curved into an incomplete ring, deficient above and behind, which represents the orifice of the cartilaginous meatus. From this point its margins gradually converge to form the narrow strip of cartilage the inner end of which is firmly attached to the rough outer extremity of the bony meatus. At the outer end of the canal, then, the anterior wall, floor, and part of the posterior wall are formed of cartilage; but as it extends inward, the cartilaginous plate becomes narrower until at its junction with the osseous meatus only the floor is formed of cartilage. The cartilaginous canal is rendered less rigid by two—sometimes three—fissures, passing through its anterior wall. These fissures, more or less vertical in direction, and transverse to the long axis of the canal, are called the fissures of Santorini. They render the canal more pliable and lessen the amount of trauma in certain surgical operations requiring great displacement of the auricle. The general direction of the membrano-cartilaginous canal is inward and somewhat upward and backward.

Fig. 4.—Auriculo-meatal cartilage, showing narrow strip of cartilage entering into the formation of the membrano-cartilaginous meatus.

Development of the Osseous Meatus.—The external auditory canal presents marked differences in the adult and the infant at term. In order to obtain a clear view of the anatomy of the conducting apparatus, it is necessary to refer briefly to the development of the temporal bone, and trace certain changes which it undergoes between birth and the middle of the fourth year, when it attains essentially the adult type. At birth the temporal bone is separable into three distinct parts,—the *petromastoid*, the *squamo-zygomatic*, and the *tympanic*. A fourth part, the styloid process, is cartilaginous at birth, and forms no part of the organ of hearing (see Plate I).

In the primordial, or cartilaginous, skeleton there is but one representative of the future temporal bone,—viz., the so-called cartilaginous ear-capsule, which encloses the otic vesicle and its outgrowths (cochlea and semicircular canals), and therefore constitutes the essential part of the organ of hearing. This is later converted by numerous centres of ossification (Hertwig-Mark) into the os petrosum, or petromastoid. On the outer surface of the os petrosum (Plate I, 5) is an irregular depression

PLATE I.

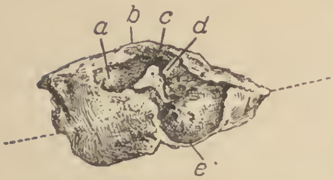


FIG. 5.—Petromastoid: *a*, antrum; *b*, tegmen tympani; *c*, tympanic vault; *d*, incus; *e*, tympanic floor.

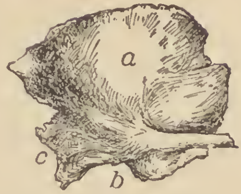


FIG. 6. — Squamozygomatic, outer surface: *a*, superior plate; *b*, Rivinian notch; *c*, inferior plate.

FIG. 7.—Right tympanic ring, outer surface.

FIG. 8. — Squamozygomatic, inner surface: *a*, inner horizontal plate (which forms part of the floor and the mid-cranial fossa); *b*, Rivinian notch.

FIG. 9.—Temporal bone: *a*, squamozygomatic; *b*, tympanic ring; *c*, petromastoid bone, posterior half; *d*, inner tympanic wall; *e*, Rivinian notch. This figure shows the relatively enormous size of the infant tympanic cavity which is practically of the same size as in the adult.

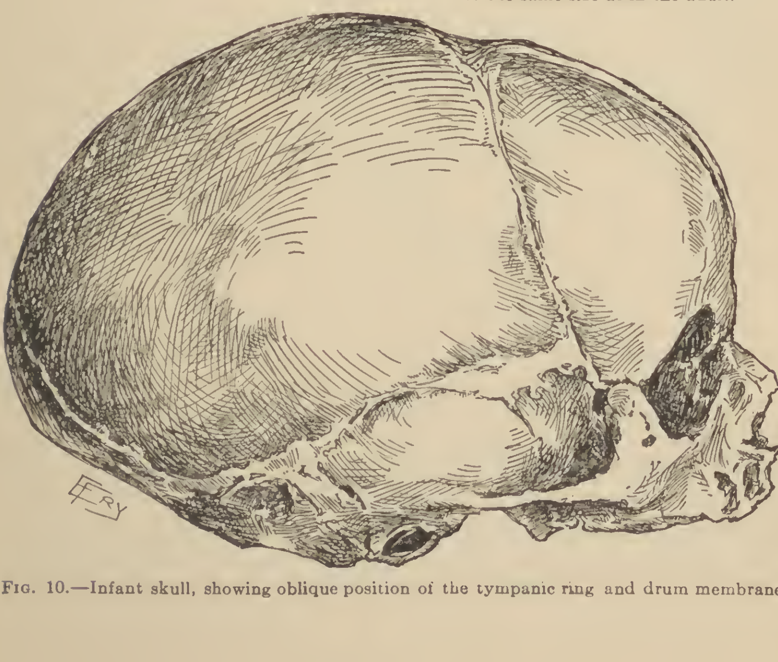


FIG. 10.—Infant skull, showing oblique position of the tympanic ring and drum membrane.

or concavity, which represents the cavity of the middle ear,—its boundaries constituting the inner wall, floor, roof, and also parts of the anterior and posterior walls of the tympanum. The tympanic cavity is completed by the squama and tympanic ring, which, with the drum membrane, form what in the adult becomes the outer tympanic wall.

The tympanic ring and squamozygomatic portion of the temporal bone have no developmental relation to the cartilaginous cranium, being developed, each from a single centre of ossification (Heisler), from the connective tissue of its enveloping membrane. The tympanic ring (Plate I, 7) at birth is a small, somewhat flattened ring of bone, which is deficient in its upper and anterior eighth. Its inner concave margin presents a groove (sulcus tympanicus) for the attachment of the tympanic membrane, or membrana tensa. The tympanic ring is applied against the outer surface of the os petrosum, shutting in the lower and outer part of the infant middle-ear cavity. Firm osseous union occurs during the first year of life. The squamozygomatic (Plate I, 6) in the new-born infant is a flat plate of bone which externally is divided into two surfaces by the small zygomatic process. Above the zygoma is the shell-like lamina of bone which forms part of the outer wall of the mid-cranial fossa. Below the zygoma, the squama is formed of two plates, an outer and an inner. The outer plate, passing downward and somewhat inward from the level of the zygoma, forms the outer wall of the tympanic vault. Its lower border is concave, and, by articulation with the two extremities of the tympanic ring, completes the bony circle surrounding the drum membrane. The bony ring thus formed is of irregular outline. That part of it formed by the lower margin of the outer plate of the squama is ungrooved and does not give attachment to the membrana tensa, or drum membrane proper. It represents the arc of a small circle, and is known as the *Rivinian segment* (Plate I, 9, *e*). The tense membrane (membrana propria) is attached only to the grooved concave margin of the tympanic process and between its upper margin and the lower margin of the squama is a space known as the *Rivinian notch* (9, *e*). This space in the living subject is closed by the membrana flaccida, or Shrapnell's membrane, to be described later.

The inner, or horizontal, plate of the squama bends inward at about the level of the zygoma, and, by articulation with the tegmen tympani, enters into the formation of the floor of the middle fossa of the skull (8, *a*).

The articulations of the bones which unite to form the infant temporal bone are clearly indicated by the illustrations of Plate I. Fig. 9 shows the temporal bone of an infant a few weeks old. It does not, however, correctly indicate its position when in articulation with the other bones of the cranium. This is better shown by Fig. 10, in which the drum membrane is shown foreshortened by its very oblique position. In fact, so nearly do the tympanic ring and drum membrane approach the horizontal plane at birth that they seem to constitute the inferior, rather than the outer, wall of the tympanic cavity at this stage of tympanic development.

The external auditory canal and drum membrane of the infant at term present the following differences from the adult type: (1) There is no bony

meatus; the drum membrane is, therefore, not protected by its position at the fundus of a bony canal, but lies in the same plane as the outer inferior surface of the skull. Its position more nearly approaches the horizontal plane than in the adult. (2) The entire external canal is membrano-cartilaginous; its direction is outward and upward, so that the drum membrane and the roof of the meatus are nearly in the same plane. The floor of the canal is in contact with the roof. These facts have a practical bearing upon the examination of the ears of infants, it being necessary by downward traction of the lobe to draw the inferior wall away from the roof, and change the direction of the canal so that the drum membrane may be brought into view.

Post-natal Changes.—Shortly after birth the following changes are inaugurated: By deposition of new bone upon the outer surface of the tympanic process or ring, this bone is converted into a bony trough, which forms the anterior wall, floor, and greater part of the posterior wall of the bony canal. That the conversion of the tympanic ring into a partial canal is effected solely by deposition of new bone upon its outer surface is evidenced by the position of the sulcus tympanicus, which remains unchanged,—*i.e.*, at the inner margin of the canal. Coincidently with these changes in the tympanic ring, the roof of the osseous meatus is formed by

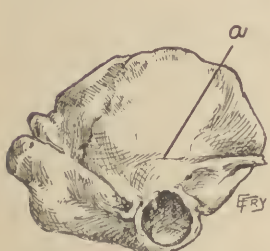


Fig. 11.—Infant temporal bone.

Fig. 12.—Adult temporal bone.

the following changes in the squama: The outer plate is gradually bent inward at the level of the zygoma, thus forming an angle with the superior, parietal plate and finally in the adult bone assuming a position nearly, but not quite, horizontal. That the superior wall of the bony meatus results largely from this bending of the outer plate of the squama, rather than from massed deposition of bone at any particular point, is shown by the changed relation of the posterior root of the zygoma, which at birth is 5 to 6 mm. above the upper margin of the tympanic orifice, and in the adult marks the upper boundary of the bony meatus (Figs. 11 and 12, *a*).

These changes progress with remarkable rapidity, so that early in the second year of life the drum membrane is located at the fundus of a short, incomplete, but well-defined canal. Coincident with the development of the bony canal, changes quite as marked are taking place in the membrano-cartilaginous meatus. As the tympanic ring is built out into a bony trough, the anterior wall, floor, and posterior wall of the membranous meatus are drawn downward and outward, becoming adherent as a membrano-cutaneous lining to the walls of the osseous canal. The floor of the membranous meatus is, therefore, no longer in contact with the roof. At the end of the first year, the interior of the meatus in its natural position no longer presents the appearance of a transverse slit as at birth, but of a tube with a well-defined lumen.

The Osseous Meatus.—In the adult the bony canal presents fairly constant characteristics. Its four walls—*i.e.*, the superior, anterior, inferior, and posterior—differ in structure, conformation, and length. The superior wall, or roof, is formed of the outer plate of the squama, its direction from without being inward and slightly downward. The anterior wall and floor are formed of very dense bone, and developed wholly from the tympanic ring. They are markedly convex in the direction of the long axis of the canal. The greatest convexity of both the anterior wall and floor is found at a point nearer the inner than the outer extremity of the canal. This is the narrowest part of the bony meatus and is called the *isthmus*. Beyond the isthmus, the floor dips strongly downward to the point of attachment of the inferior margin of the drum membrane, forming a depression, or sulcus, in which foreign bodies if small may be hidden from view. The posterior wall is composed in part of the dense tympanic plate, and partly of the premastoid plate from the outer lamella of the squama.

A consideration of the relative length of the different walls of the canal helps one to appreciate the oblique position of the drum membrane. The anterior wall is longer and projects 5 or 6 mm. further inward than the posterior wall, and the floor projects further inward by 5 or 6 mm. than the roof. As the inner, terminal margins of these walls merge into the elliptical frame or groove which supports the drum membrane, it is obvious that the latter occupies a plane which forms an obtuse angle with the superior and posterior walls, and an acute angle with the anterior wall and floor. The drum membrane is, therefore, not placed vertically or at right angles to the long axis of the canal, its outer surface looking outward, downward, and forward.

Politzer gives the average length of the four walls of the bony canal as follows: superior wall, 14 mm.; inferior wall, 16 mm.; posterior wall, 15–16 mm.; and anterior wall 17–18 mm. Some years ago the author made careful measurements of a series of bones with a view of determining the length of the postero-superior canal wall,—*i.e.*, taken from the annulus tympanicus internally to the spine of Henle externally,—and found variations between 12 and 18 mm., the average being 14.5 mm. Obviously the length of the bony canal is subject to very considerable

variations in different individuals. Cross section of the outer third of the bony meatus shows it to be of rather circular form, whereas internally—*i.e.*, beyond the isthmus—it becomes elliptical.

The external auditory canal as a whole presents great variations in different individuals. Some are wide and sufficiently straight to allow easy inspection of the drum membrane, while others are narrow and present curves necessitating considerable manipulation in order to bring the lower half of the drum membrane into view. The length varies from 1 to 1½ inches, 1¼ inches being about the average. Of this the bony canal forms rather less than half. The membrano-cartilaginous portion is somewhat curled upon its long axis, and is rather wider in its central portion than at its orifice in the concha or at its junction with the bony meatus. The long axes of the two portions of the canal are not in the same straight line, the membrano-cartilaginous part being directed inward and slightly upward and backward, and the osseous canal inward and slightly downward and forward. It is often necessary, therefore, in order thoroughly to inspect the drum membrane, to draw the auricle upward and backward, thus bringing the two portions of the canal into the same straight line and the lower portion of the membrana tympani into view.

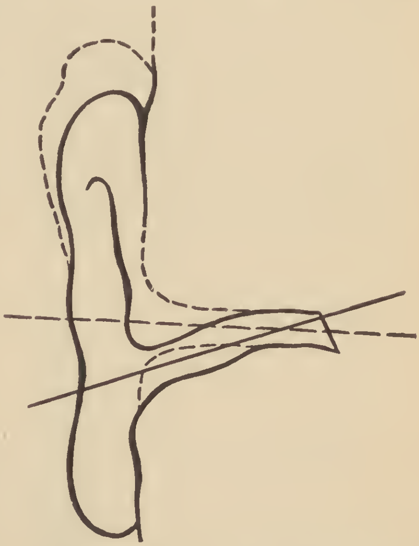


Fig. 13.—Diagram: dotted lines showing influence upon direction of canal of drawing auricle upward and backward.

The integument lining the auditory canal is continuous externally with that of the concha, and internally with that covering the outer surface of the drum membrane. The skin covering the drum membrane and canal seems to grow eccentrically from a point at the centre of the membrana tympani, light substances—*e.g.*, small disks of paper—attached to any part of the drum membrane being found to travel first to its periphery and thence along the wall of the canal to the concha. The integument is much thicker in the membrano-cartilaginous than in the bony meatus. In the membranous canal it is from 1 to 1½ mm. in thickness, and contains hair-follicles, sebaceous glands, and numerous ceruminous glands. The latter are arranged thickly in the posterior wall and floor. The ducts of the ceruminous glands may open either directly into the auditory canal or into the hair-follicles.

The Tympanum.—The tympanum (tympanic or middle-ear cavity) is the name applied to the bony space which contains the ossicular chain.

Its outer wall is composed largely of the drum membrane, and but for this structure it would be open to the outside air by way of the external auditory canal. It does communicate with the nasopharynx by means of the Eustachian tube. The roof of the tympanum lies on a plane considerably higher than the roof of the bony meatus, and its floor lies below the level of the floor of the bony canal. This fact has led to its being considered as composed of three parts,—namely:

(1) The *vault* (attic, epitympanic space), or that portion of the tympanic cavity lying above the level of the short process of the malleus.

(2) The *atrium*, or that portion of the middle-ear cavity the upper and lower boundaries of which are horizontal lines, or planes, passing through the upper and lower margins of the membrana tensa.

(3) The *hypotympanic space*, or that part lying below the level of the floor of the bony canal (Fig. 14).

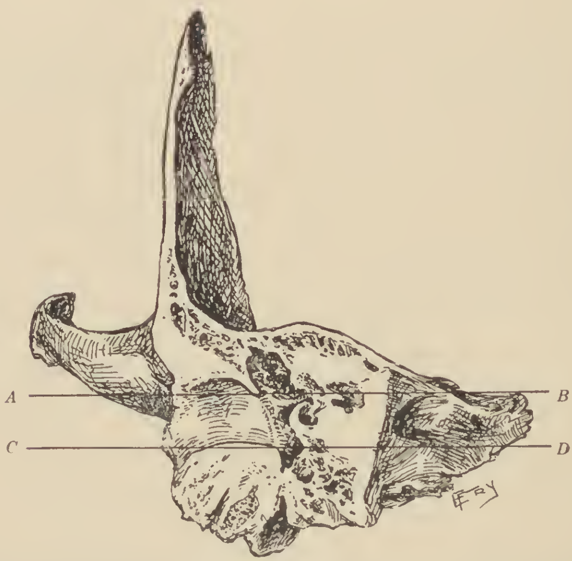


FIG. 14.—Vertical section through tympanic cavity and bony meatus. Above line A–B, tympanic vault. Between lines A–B and C–D, atrium. Below line C–D, hypotympanic space.

The tympanum, or middle ear, viewed as a whole, is an irregularly wedge-shaped cavity, lying for the most part between the outer surface of the petrous bone and the drum membrane. Its greatest diameter is the vertical; its smallest is from without inward,—*i.e.*, from the drum membrane to the inner tympanic wall. Its roof corresponds in position with the floor of the middle cerebral fossa; its floor is in relation with the jugular fossa which lodges the bulb of the jugular vein. The roof is much wider—from without inward—than the floor, which measures but 2 to 3 mm. The gradual convergence from above downward of the outer and inner

walls brings the cavity of the tympanum somewhat into the form of a wedge, the edge of which—directed downward and inward—corresponds with the tympanic floor.

The tympanic cavity presents six walls, which call for careful study.

The Inner Wall (Fig. 15).—The inner wall of the tympanum is formed by the outer surface of the petrous bone. It presents the following important landmarks: At a variable distance above the mid-point between the roof and floor is a well-marked ridge running from before backward (Fig. 15, *a*), and forming a natural boundary line between the inner wall of the vault and the inner wall of the atrium. This is formed by the outer wall of the Fallopian canal, which lodges the facial nerve. Just below the posterior half of the facial ridge is an oval depression, the fossa ovalis, at the bottom of which the *oval window* (Fig. 15, *b*) is plainly visible. The long diameter of the oval window is horizontal. It leads inward and

Fig. 15.—Inner tympanic wall (drum membrane and outer bony wall of tympanic vault removed).

somewhat backward into the central cavity of the bony labyrinth (vestibule). In the living subject it is closed by the foot-plate of the stapes. Below the oval window the inner wall bulges into the cavity of the atrium, forming a rounded eminence, convex from above downward, called the *promontory*. This prominence is formed by part of the first turn of the cochlea. In form it resembles roughly the lateral half of a cone divided longitudinally from apex to base, the apex being directed forward and the base backward. Immediately behind the promontory, and below and somewhat behind the oval window, is another depression, the niche of the round window (15, *d*). From this niche a somewhat circular orifice, the fenestra rotunda, leads forward and inward into that portion of the spiral tube of the cochlea known as the scala tympani. The round window is closed by

a delicate membrane which has received the name *membrana tympani secundaria* (Scarpa). Again comparing the promontory to a divided cone, we find that the apex is lost near the junction of the inner and anterior walls, or rather where the anterior wall merges into the funnel-shaped orifice of the Eustachian tube (*e*). Just above the Eustachian tube is the canal for the tensor tympani muscle. That portion of the tensor tympani canal presenting upon the inner wall of the tympanum is usually incomplete (15, *f*). It passes obliquely upward and backward above the promontory to a point in front of the anterior end of the oval window. Here it terminates in a thin lamella of bone, curled somewhat outward (processus cochleariformis), around which the tendon of the tensor tympani turns to pass outward and downward to its attachment to the hammer handle. Passing now to the vault, we find just above and parallel with the posterior end of the facial ridge a linear elevation of the inner wall. This elevation marks the position of the external or horizontal semicircular canal. It occurs not as a distinct ridge, but simply as a longitudinal bulging of the inner wall at this point (15, *c*). It is characterized chiefly by the smooth and ivory-like density of the bone in this situation.

THE SUPERIOR WALL (Fig. 16).—The roof of the tympanum is of surgical interest chiefly from the fact that its upper surface forms part of

FIG. 16.—Tympanic roof.

the floor of the middle cerebral fossa. It is of varying thickness in different bones, is often exceedingly thin, and occasionally exhibits defects of continuity (apertures), in which case the cavity of the middle ear is separated from the middle fossa of the skull only by loose connective tissue. The plate of bone forming the roof is a direct continuation of that covering the superior surface of the petrous bone. Externally it is united to the

inner horizontal plate of the squama, forming the petrosquamosal suture, which is plainly visible in infancy and occasionally persists in adult life. A statement repeated in several text-books is to the effect that the inner horizontal plate of the squama enters into the formation of the tympanic roof. This statement is not correct. In any temporal bone in which the petrosquamosal suture can be recognized, it will be found that, if we make a vertical perforation downward through this suture, the instrument will appear below in the bony meatus and not in the tympanic cavity. The roof of the tympanum is formed, therefore, wholly by the superior plate of the os petrosum.

The Anterior Wall (Fig. 17).—The upper and lower limits of the anterior wall of the tympanum are not clearly defined. Superiorly it merges by a gentle curve into the roof, and below it presents no angle of junction or natural landmark separating it from the tympanic floor. Internally and externally its limits are clearly defined by the inner and outer walls. It presents two prominent landmarks,—namely, the mouth of the Eustachian tube and the canal for the tensor tympani muscle. Two or three

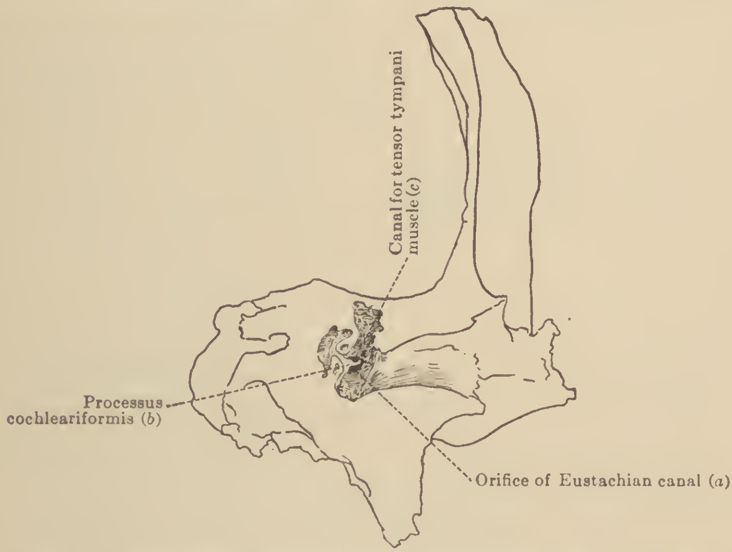


Fig. 17.—Vertical section of temporal bone, showing anterior wall of tympanum.

mm. above the level of the tympanic floor is the tympanic orifice of the Eustachian tube (Fig. 17, a). The length of this bony canal averages about 12 mm. Its direction from the tympanum is forward, inward, and downward. Just above the Eustachian orifice is the beginning of the tympanic portion of the canal for the tensor tympani muscle (c), which has already been described in connection with the inner tympanic wall. This canal begins at the base of the skull in the anterior part of the petrous bone. In its passage through the petrous bone it lies just above and internal to

the Eustachian canal, from which it is separated by a thin lamella of bone. The lower portion of the anterior tympanic wall is in relation to the canal for the internal carotid artery. In this situation the bony plate separating the middle-ear cavity from the artery is in some bones exceedingly thin.

The Posterior Wall (Fig. 18).—The posterior wall presents two portions which must be examined separately, — namely, the posterior wall of the vault and the posterior wall of the atrium. The posterior wall of the vault presents centrally a large, irregularly triangular opening the base of which is directed upward and the apex downward. This triangular opening is known as the aditus ad antrum, and marks the dividing line between the tympanic vault and the so-called mastoid antrum.

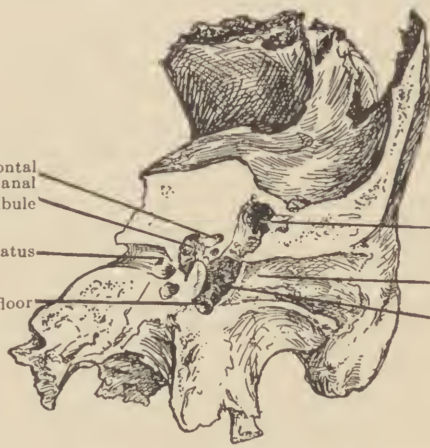


Fig. 18.—Section showing posterior wall of tympanum.

The antrum in reality is nothing more than a posterior prolongation of the vault. The posterior wall of the atrium is narrow from within out, its greater diameter being the vertical. Its chief landmark is a small pyramidal process (processus pyramidalis) which projects forward into the atrium a little above the mid-point between the round and oval windows. It incloses a canal which contains the stapedius muscle, and presents at its apex a small opening through which the stapedius emerges to pass forward to its attachment to the neck of the stapes.

The Inferior Wall (Fig. 19).—The floor of the tympanum is a narrow space inclosed between the lower segment of the annulus tympanicus externally and the inner wall of the hypotympanic space internally. Viewed from above downward, it appears as a narrow depression, 5 to 7 mm. long and 2 to 3 mm. wide, containing more or less cancellous or diploic tissue. This diploic tissue throws into clear relief the hard, compact bone of the floor of the osseous canal, which forms its outer boundary. The floor of the tympanum is in relation anteriorly with the canal for the internal carotid artery, posteriorly with the jugular fossa. The bony plates separating the hypotympanic space from the artery in front and

the bulb of the jugular vein behind are sometimes exceedingly thin. Moreover, cases have been observed in which, as a congenital defect or as a result

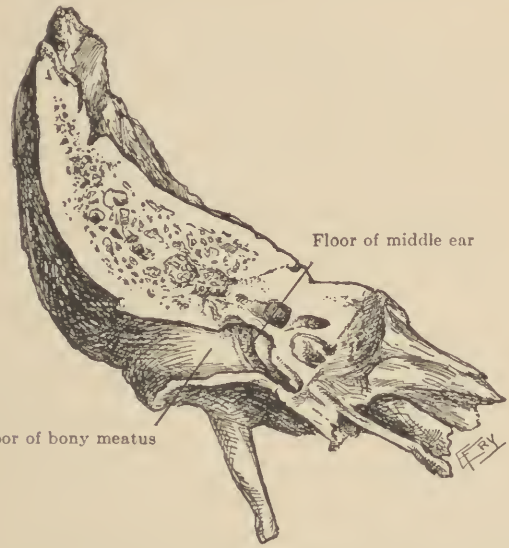


Fig. 19.—Horizontal section through tympanum, showing tympanic floor.

of necrosis, defects in the tympanic floor have allowed one or the other of these vessels to protrude into the lower part of the tympanic cavity,—a fact which, though exceedingly rare, should be borne in mind in carrying out any surgical measures in this region.

The External Wall.—It now remains to be explained how this irregular but perfect little cavity is closed externally. The outer wall is

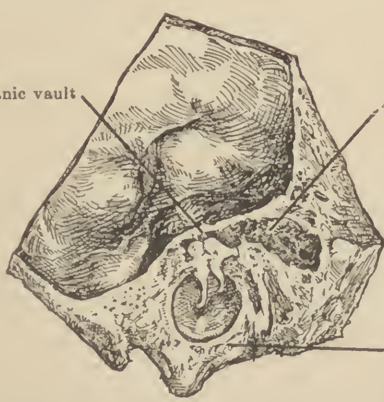


Fig. 20.—Inner, or tympanic, view of drum membrane.

formed chiefly by the membrana tympani; but, since the middle ear extends in all directions somewhat beyond the limits of the drum membrane,

there is a peripheral frame of bone which must be accounted for as part of the outer wall. This in the atrium and hypotympanic space consists of the inner surface of the tympanic ring. In the vault the outer wall is formed of the thickened inner edge of the outer inferior plate of the squama. This presents usually a fairly compact surface of bone (Fig. 20).

The Annulus Tympanicus.—The inner margin of the bony meatus forms an irregular ring (annulus tympanicus), to which the drum membrane is attached. Owing to inequalities in the lengths of the different walls,—the floor and anterior wall being longer and extending further inward than the roof and the posterior wall,—this ring is irregularly ovoid or elliptical in shape, the long axis of the ellipse being from above downward and forward. These peculiarities also explain the oblique position of the drum membrane, which is not at right angles to the long axis of the bony meatus but forms decidedly obtuse angles with its roof and posterior wall.

The Drum Membrane.—The membrana tympani is the strong lamina of fibrous tissue which forms the outer boundary of the middle ear. It is covered externally by integument continuous with that lining the external auditory canal, internally by mucous membrane continuous with that covering the tympanic walls. Centrally it is drawn inward by the lower end of the hammer handle, to which it is attached, so that its outer surface is concave. It is attached peripherally to the concave margin of the annulus tympanicus, so that it necessarily assumes the oblique position of that bony ring, its outer surface looking outward and also strongly downward and forward. So intimately is it connected with certain parts of the malleus that it is impossible to give any practical description of the drum membrane without assuming some knowledge of the anatomical peculiarities of that little bone. We shall pause, therefore, in order to describe briefly the ossicular chain.

The Ossicles.—The auditory ossicles are a system of little articulated bones, three in number, which connect the drum membrane with the labyrinth. The malleus, placed externally, is directly connected with the drum membrane; the stapes, the smallest of the three, presents a flattened bean-shaped surface which fits into and closes the oval window; and the incus, or middle bone, articulates externally with the malleus and internally with the stapes, thus forming a chain of movable levers which responds easily to the slightest movements of the drum membrane and transmits all sonorous impressions through the oval window to the labyrinth and end-organs of the auditory nerve.

The MALLEUS, or hammer (Plate II, 22), is the only one of the ossicles which is in direct contact with the drum membrane, and is therefore the only one which can invariably be seen through the auditory canal. The *head* (22, A) is the upper, rounded portion which articulates with the body of the incus. It is smooth in all its aspects except the posterior, which presents an irregular surface for articulation with a rough depression on the anterior surface of the incus. It terminates below in a constricted

PLATE II.

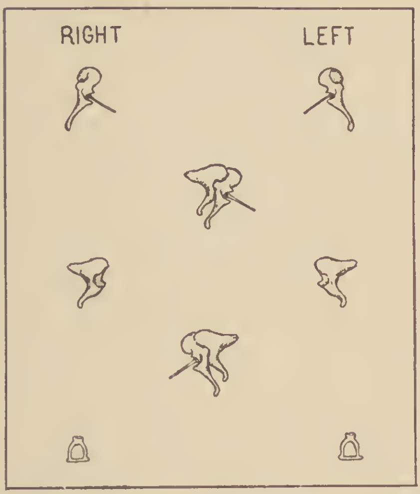
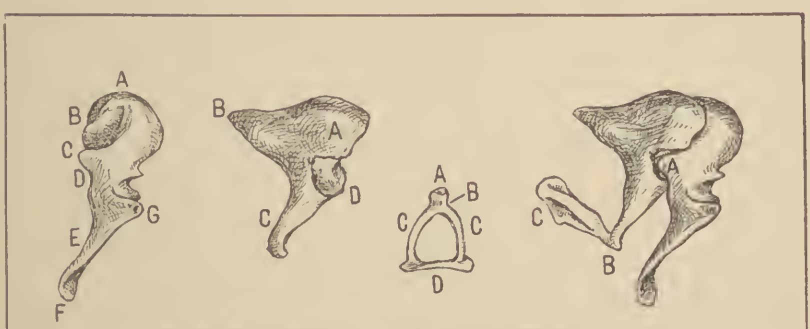


FIG. 21.—Ossicles, right and left, only slightly enlarged above actual size.



22 23 24 25

FIG. 22.—Malleus, enlarged; *A*, head; *B*, articular process; *C*, cog-process; *D*, neck; *E*, hammer handle; *F*, umbo, *G*, short process.

FIG. 23.—Incus; *A*, body; *B*, short process; *C*, long arm; *D*, cog-process.

FIG. 24.—Stapes; *A*, head; *B*, neck; *C,C*, crura; *D*, foot-plate.

FIG. 25.—Ossicles in articulation; *A*, cog-processes in apposition; *B*, incudo-stapedial joint; *C*, foot-plate.

portion, the *neck* (22, D). Passing downward and backward from the neck is a narrow shaft of bone called the hammer handle, or *manubrium mallei* (E). The handle presents two rather sharp edges,—an external edge attached to the drum membrane, and an inner edge directed toward the inner tympanic wall. These two edges are separated by two comparatively broad surfaces which are directed forward and backward respectively. At its lower extremity the hammer handle broadens into a somewhat flattened surface of bone called the *umbo* (F). At the beginning of the external margin of the hammer handle, or rather at its junction with the neck, is a short, pointed process of bone, directed outward and somewhat forward and upward, the *short process* (G). This also is in contact with the drum membrane, which it pushes before it into the cavity of the auditory canal. As viewed by reflected light through the auditory meatus, it appears as a small, whitish glistening projection in the upper anterior quadrant of the membrana tympani, and constitutes one of the most constant and useful landmarks. From the anterior surface of the neck, a slender spiculum of bone passes forward and slightly downward, its anterior extremity lodging in the Glaserian fissure (see mallei in small figures, Plate II, 21). This is the long process of the malleus, or processus folianus. It is always present at birth, but is usually absorbed later in life. Its position is identical with that of the anterior ligament of the malleus, which usually surrounds it.

The INCUS (Plate II, 23) is the central link of the ossicular chain. It consists of a body and two processes. The body (23, A) with the short process (B) forms an irregular cone, flattened from without inward, and presenting on its anterior surface, or base, a rough depression for articulation with the head of the malleus. Posteriorly the body terminates in a somewhat pointed extremity, the short process of the incus (23, B). Extending downward from the anterior inferior corner of the body, and almost at right angles with its long axis, is a slender shaft of bone, the *long arm* (23, C). When the ossicles are in normal position, this process extends downward and backward, and is almost parallel with the manubrium mallei. Its lower extremity hooks sharply inward, and terminates in a rounded surface for articulation with the head of the stapes. This is known as the lenticular process.

The STAPES (Plate II, 24) is the smallest and—functionally at least—the most important of the three ossicles. It resembles almost perfectly a stirrup in form, and consists of a head (A), neck (B), two crura (C), and a bean-shaped plate of bone, the foot-plate (D), which fits into and closes the oval window. The foot-plate varies somewhat in size and shape in different individuals to conform to variations in the size and shape of the oval window. The arms, or crura, arise from either end of the outer surface of the foot-plate, and gradually converge to meet in a small button-shaped process, the head. Between the head and the point of junction of the two crura is a circular constriction, the neck. Upon the outer surface of the head is a very slight depression, or facet, which receives the lenticular

process of the incus. When the foot-plate is in its normal position within the oval window, the whole ossicle is somewhat depressed within the oval niche, the head extending but slightly into the cavity of the atrium.

We are now in a better position to study the structural peculiarities of the membrana tympani, which the accompanying diagram may help to make clear.

As stated in a foregoing paragraph, the drum membrane is composed of three layers,—an external cuticular, a middle fibrous, and an internal layer of mucous membrane. The outer, cuticular layer is easily separated from the underlying fibrous membrane, as is frequently shown by its early exfoliation when the drum membrane is the seat of an acute inflammation. The internal, mucous layer, on the other hand, is so closely adherent as to be practically inseparable.

The drum membrane as a whole is divisible into two parts,—(1) the tense membrane, or membrana propria; and (2) the flaccid membrane, or Shrapnell's membrane.

The membrana tensa forms the outer wall of the atrium. It is attached peripherally to the sulcus tympanicus, or that portion of the annulus tympanicus which represents the original auditory process, or tympanic ring. Toward the upper and anterior pole of the drum membrane is seen in the living subject a small knob-like projection. This is the short process of the malleus (Fig. 26, D). Extending downward and backward from

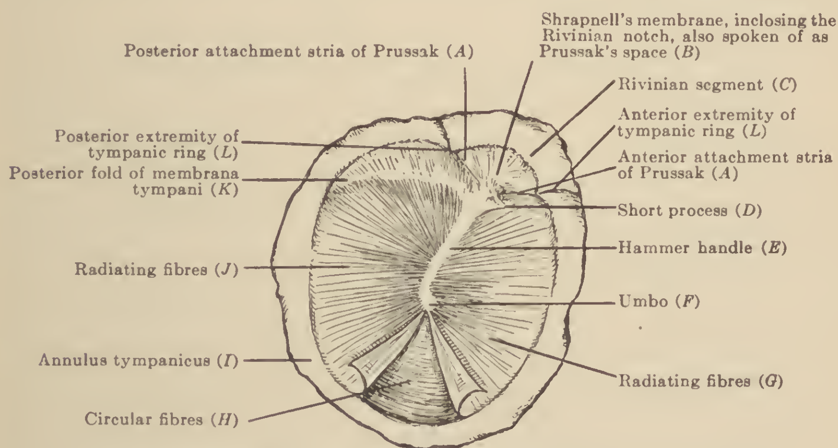


Fig. 26.—Diagrammatic picture of the membrana tympani and its attachments.

the short process to the centre of the drum membrane is a line showing the position of the hammer handle (26, E), the lower terminal expansion of which is known as the umbo (F). The middle fibrous layer of the drum membrane is confined to the membrana tensa. It is composed of two layers,—an outer layer of radiating fibres (G, J), and an inner layer of circular fibres (H). The outer, radiating fibres are attached centrally to

the umbo and to some extent also to the lower half of the hammer handle, from which central attachment they radiate toward the periphery of the membrane. They are therefore more closely arranged at the centre, and for this reason appear thicker and more numerous here than at the periphery. The inner circular fibres are arranged in concentric circles about the umbo. They are very sparsely distributed toward the centre of the membrane, but are much more numerous in the outer circles. In the upper part of the membrane they cross the radiating fibres in considerable numbers. The circular fibres are said to collect at the periphery and, together with certain cartilage-cells and tough connective-tissue fibres, aid in forming the thickened peripheral margin (annulus tendinosus) which is inserted into the tympanic ring.

The membrana propria (M. tensa) is inserted into the tympanic groove throughout the entire extent of the original tympanic ring. Between the anterior and posterior extremities of the tympanic ring (spina tympanica major and spina tympanica minor), the upper margin of the membrana tensa has no bony attachment except to the short process of the malleus. In the living subject this upper margin is sometimes indicated by two delicate lines radiating from the short process, one leading forward to the spina tympanica major and the other leading upward and backward to the spina tympanica minor. These two lines (A, A) are known as the attachment striæ of Prussak. Between Prussak's striæ below and the Rivinian segment above, is a small, somewhat fan-shaped space known as the Rivinian notch (B). This space is inclosed by the membrana flaccida (Shrapnell's membrane), next to be described. Below the posterior stria of Prussak, and more horizontal in direction, is a prominent fold of the drum membrane, always easily discernible, known as the *posterior fold* (K). It passes backward from the short process to the tympanic ring.

Shrapnell's Membrane.—The Rivinian space, or notch, is closed by a membrane which is thinner and less tense than the membrana tensa; hence the name, *membrana flaccida*. It consists chiefly of an outer cutaneous layer continuous with the integument covering the membrana tensa and the roof of the bony meatus, and an inner layer of mucous membrane continuous with that lining the tympanic cavity. Between these there are a few interlacing fibres of delicate connective tissue, but there is no distinct layer of fibrous tissue. It is in reality a very small membrane which is sometimes difficult to outline in the living subject: that is to say, there sometimes seems to be no demonstrable space between the striæ of Prussak and the inner margin of the roof of the bony meatus (incisura Rivini). Shrapnell's membrane forms part of the outer wall of the vault, and measures, according to Politzer, 1½ mm. vertically and 2 mm. transversely,—*i.e.*, from before backward.

The Ossicles, their Articulations and Relative Positions within the Tympanum (Fig. 27).—Before going further it is essential that the student should have a clear understanding of the position within the middle ear of the different parts of the ossicular chain.

The ossicles are lodged partly in the vault and partly in the atrium. In the vault are found the head of the malleus and the body of the incus. The remaining parts of the ossicular chain—*i.e.*, the short process and handle of the malleus, the long arm of the incus, and the stapes—are located within the atrium.

The Malleus.—The head of the malleus is lodged in the anterior part of the vault. Posteriorly it presents a rough surface for articulation with the incus. The neck of the malleus is on a level slightly below the inner margin of the roof of the bony meatus. Just below the outer ante-

Fig. 27.—Temporal bone with drum membrane and outer wall of vault removed, showing relative position and ligaments of the ossicles.

rior surface of the neck, the short process projects into the cavity, or lumen, of the bony canal, pushing the drum membrane before it. An imaginary horizontal line tangent to the upper surface of the short process (Fig. 27, AB) marks the boundary line between the atrium and vault. Extending downward and backward from the short process is the hammer handle, or manubrium mallei. Its sharp external margin is connected with the drum membrane by means of a thin interposed layer of cartilage.

Ligaments of the Malleus.—The head of the malleus is attached to the under surface of the tegmen tympani by a thin suspensory ligament. From the anterior surface of the neck and from the long process (processus folianus) a short, thick, fibrous band, the anterior ligament, passes forward to the spina tympanica major and Glaserian fissure, to which it is attached. It surrounds the long process which is commonly absorbed in later life. The posterior ligament seems really to be a posterior fasciculus of the lateral ligament. It extends backward from the posterior surface of the neck to be attached to the outer wall of the vault. These two ligaments,—the anterior and posterior,—though not quite in the same straight line, are sometimes spoken of as the rotary axis band of the

malleus. They prevent lateral displacement of the malleus, and account for the constancy of the short process as a landmark of the drum membrane: the short process may change its position—*i.e.*, revolve—as the malleus rotates about its axis band, but its location remains practically unchanged. The external ligament (ligamentum laterale) is attached to the outer surface of the neck above the short process, and spreads out, fan-shaped, to be attached to the outer bony wall of the vault.

The Incus.—The body of the incus occupies the posterior half of the vault. Its posterior conical extremity, the short process, is lodged in a small depression, or facet (fossa incudis), in the posterior wall of the vault just below the aditus, or opening into the antrum. Both the fossa incudis and the articular surface of the short process are covered with thin layers of cartilage. The joint is held together by a strong capsular ligament and also by bands of fibrous tissue springing from the periosteum of the adjacent bone surfaces. Like the malleus, the incus is supplied with a suspensory ligament connecting its superior surface with the tympanic roof (Brühl)[1]. The anterior surface of the incus articulates with the head of the malleus.

Articulation of the Malleus and Incus (Plate II, 25).—In describing this joint it is necessary to allude once more to certain points in the anatomy of the bones. The posterior surface of the head of the malleus presents an irregularly oblong and spiral depression, or groove, extending from above downward and inward to the neck. The lower end of this groove is bounded externally by a projecting point of bone which exerts inward pressure upon a somewhat similar point of bone on the incus. This is called the cog, or tooth-process, of the malleus (Plate II, 22, C). On the anterior surface of the body of the incus is a spiral ridge which conforms to, and is received into, the spiral groove of the malleus. Internal to the lower end of the incudal ridge is a triangular projection of bone (23, D), which is spoken of as the cog, or tooth-process, of the incus. When the bones are in normal articulation, the tooth-process of the malleus is in contact with that of the incus (Plate II, 25). The result of this mechanism is that when the malleus rotates inward,—*i.e.*, when the hammer handle moves inward,—the incus is made to execute a similar inward rotation; when, however, the malleus is forcibly rotated outward,—as in sudden forcible condensation of the air in the tympanum, sneezing, Valsalva inflation etc.,—the cog, or tooth-process, of the malleus separates from that of the incus and the joint opens, so that excessive outward excursions of the hammer handle are not necessarily accompanied by corresponding excursions of the long arm of the incus. By this provision, the stapes is guarded against the influence of extensive outward movements of the drum membrane which might otherwise result in dislocation of the

[1] Brühl states that there are distinct suspensory ligaments connecting both the malleus and incus with the tympanic roof. As there is a difference of opinion, however, as to the usual presence of a suspensory ligament of the incus, such a ligament is not included in Fig. 27, the author himself never having seen it.

stapes from the oval window. The opposed articular surfaces of the malleus and incus are lined with cartilage. The joint is provided with a strong capsular ligament and is further strengthened by strong fibrous bands.

The *long arm of the incus* passes downward and backward into the cavity of the atrium. It is behind and more or less parallel with the hammer handle, but is not in contact with the drum membrane. It is shorter than the hammer handle, usually terminating about the mid-point of the latter, or even at a higher level.

The Incudo-stapedial Joint is in the upper and posterior part of the atrium (see Fig. 27, and Plate II, 25). The rounded lenticular process of the incus and also the depression on the head of the stapes are covered with hyaline cartilage, and are held together by a capsular ligament.

Articulation of the Stapes with the Fenestra Ovalis.—The margins of the oval window and the edges of the foot-plate of the stapes are covered with a thin layer of cartilage, which is said also to line the vestibular surface of the latter. These surfaces are held together by an annular ligament consisting of elastic and connective fibres derived from the periosteum of the inner margins of the fenestra and passing to the foot-plate of the stapes. The annular ligament is much wider in front than behind.

So far as the writer knows, the ossicular chain is the only system of movable joints in the body the movements of which are not under the control of voluntary muscles. Aside from their minute size, the articular structures of these joints are the same in kind as are found in connection with the larger bones elsewhere. The movements of the ossicles, however, are initiated and maintained altogether by forces originating outside the body,—*i.e.*, aerial vibrations set in motion by some vibrating body. The ossicular ligaments seem, therefore, to play a particularly important rôle, for they not only hold the joint surfaces in proper apposition, but serve to limit the activity of each ossicle to that particular form of motion to execute which is apparently its sole function.

The Intra-tympanic Muscles. — The stapedius (Fig. 28) is a small muscle, about 5 or 6 millimetres long, the belly of which is enclosed in the bony canal of the eminentia pyramidalis. This canal is buried in the premastoid plate, lying in front of and parallel with the descending portion of the facial canal. Its upper extremity curves forward and appears upon the posterior wall of the tympanum as a very small bony prominence known as the *eminentia pyramidalis* (Fig. 28), the apex of which is just behind the oval window. The muscle arises from the walls of this canal, its fibres converging above into a tendon which emerges through a small orifice in its apex. From this point it passes forward to be attached to the head of the stapes and lenticular process of the incus. The canal of the pyramidal eminence communicates by a small orifice with the facial canal behind it, and through this a small branch of the facial nerve passes to supply the stapedius muscle.

The Tensor Tympani Muscle (Fig. 29)—This muscle is from 18 to 20 millimetres long. It arises chiefly from the walls of an incomplete bony canal lying just above the osseous portion of the Eustachian tube, from the superior wall of the cartilaginous part of the Eustachian canal, and also by a few fibres from the inferior surface of the petrous bone. Its

Fig. 28.—Tympanum of infant six months old. (Left.)

Fig. 29.—Tympanum of infant six months old. (Right.)

tendon passes backward and upward across the inner wall of the tympanum, and emerges from its bony canal in front of the anterior end of the oval window. Here it curves around the processus cochleariformis and passes outward to be attached to the upper part of the handle of the malleus (Fig. 29). It is supplied by the fifth nerve.

The Lining Membrane of the Tympanum.—The mucous membrane lining the tympanic cavity is directly continuous through the Eustachian tube with that of the nasopharynx. Histologically it varies in character in different parts of the tympanum. Over the promontory it is of the cuboidal variety, while on the anterior half of the floor, and in the vicinity of the tympanic orifice of the Eustachian tube, it consists of ciliated cylindrical cells (Politzer, Gruber). In the vault the cells are of the squamous variety, this type being continued into the aditus and antrum and persisting throughout the lining membrane of the mastoid cells. Beneath the epithelial, or mucous, layer are two layers of closely related connective-tissue fibres, the deeper of which is in contact with the osseous walls of the tympanum and constitutes their periosteal lining. The periosteal layer is very richly supplied with blood-vessels.

Within the atrium the mucoperiosteal lining is for the most part smooth and very closely applied to the tympanic walls. In the vault, on the other hand, the mucosa is thrown into folds and reduplications, some of which hang down from the tegmen tympani and cover the bodies of the malleus and incus.

The Pouches, or Pockets, of the Membrana Tympani.—There are three fairly constant and partially closed spaces (pockets, pouches) in relation to the drum membrane, which now must be described. The

first is a small space lying between the neck of the malleus internally and Shrapnell's membrane externally. It is bounded above by the external ligament of the malleus, internally by the neck of the malleus, below by the superior surface of the short process, and externally by the membrana flaccida. It is called PRUSSAK'S SPACE, its location being indicated upon the outer surface of the drum membrane by the boundary lines of the Rivinian notch (Fig. 26, B). Prussak's space is said to communicate, by a small aperture in the external ligament of the malleus (its roof), with the attic, or vault.

Stretching from before backward across the upper part of the cavity of the atrium, and passing between the handle of the malleus and the long arm of the incus, is a reduplication of mucous membrane which, by its attachment to the posterior surface of the neck and upper half of the hammer handle, is divided into two parts,—the anterior and posterior folds. The posterior fold is attached anteriorly to the neck and upper half of the handle of the malleus, and above and behind to the postero-superior margin of the drum membrane. Its dependent, free margin covers the chorda tympani nerve as it passes upward and forward toward the Glaserian fissure. Between this fold and the inner surface of the drum membrane is a pocket, or space, opening downward, which is known as the POSTERIOR POUCH OF THE MEMBRANA TYMPANI. The anterior fold is smaller, and extends from the neck and handle of the malleus to the antero-superior margin of the drum membrane. Between it and the inner surface of the drum-head is another, smaller space,—also opening downward,—the ANTERIOR POUCH OF THE DRUM MEMBRANE. Prussak's space is said to communicate with the posterior pouch behind, but has no communication with the anterior pouch. Both pockets of the drum membrane—*i.e.*, the anterior and posterior—open below into the cavity of the atrium.

With regard to the exact histological relations of the membranes entering into the formation of these pouches, somewhat different views have been held. Von Troltsch, who first described the anterior and posterior pockets, regarded the membrane as a true reduplication of the membrana tympani,—this view being based upon his observation of certain fibres which he believed to be identical in structure with those of the fibrous lamina of the drum membrane. Politzer appears to regard them as reduplications of the inner, or mucous, layer of the drum membrane. Gruber in his investigations failed to find any fibres characteristic of the membrana propria, and regards the reduplications in question simply as folds of mucous membrane depending from the tympanic roof.

The three spaces above described are all situated below the level of the external ligament of the malleus which forms the roof of Prussak's space. Above this level the attic is itself divided into compartments more or less completely separated from each other by folds of mucous membrane. These spaces, while less constant in their arrangement than the pockets of the membrana tympani, must nevertheless be held in mind

on account of their possible influence upon the course of a suppurative process within the vault.

The Attic Spaces.—The head of the malleus and body of the incus divide the attic into two chambers, an inner and an outer chamber. This partition is often completed by a fold of mucous membrane (the malleo-incudal fold) passing from the tympanic roof to the upper surfaces of the malleus and incus. This vertical fold incloses between its layers the suspensory ligaments of the malleus and incus, and extends from the anterior ligament of the malleus in front to the aditus ad antrum behind. The outer chamber of the attic is bounded internally by the bodies of the ossicles and the malleo-incudal fold, and externally by the outer bony wall of the vault. The outer chamber is subdivided by a horizontal fold of mucous membrane into an upper and a lower attic space. This horizontal fold is attached to the head of the malleus and body of the incus internally and to the osseous outer wall of the attic externally, and extends from the anterior ligament of the malleus in front to the short process of the incus and cella incudis behind. The lower attic space is bounded below by the ligamentum laterale mallei, which separates it from Prussak's space. The upper attic space opens behind into the aditus ad antrum (Brühl). The membranous partitions between these spaces, while usually present, probably present many variations from those above described. They are said to contain many small apertures through which fluid may pass from one enclosure to another. This subdivision of the attic into compartments adds very materially to the gravity of a suppurative process within the vault. It explains the surgical inadequacy of merely puncturing the drum membrane (paracentesis), and the importance of making a free incision through Shrapnell's membrane and the soft tissues immediately behind it in all cases of acute purulent inflammation involving the vault.

Vascular and Nervous Supply of the Tympanum.—ARTERIES.—The arterial supply of the middle ear is derived from the external and internal carotid arteries.

From the *external carotid* are derived: (1) *the tympanic branches of the ascending pharyngeal artery.* This vessel gives off several small branches which enter the Eustachian tube, supplying the tubal muscles and mucous membrane. One or more of these enter the tympanum, pass upward over the promontory, and anastomose with the tympanic branches of the middle meningeal artery. (2) *The tympanic branches of the middle meningeal artery.* After entering the skull the middle meningeal gives off a branch (the petrosal) which enters the hiatus canalis Fallopii, anastomosing with the stylomastoid, and also several small branches which enter the tympanum through the petrosquamosal suture. These latter supply the roof and part of the inner wall of the tympanum. (3) The *tympanic branch of the internal maxillary* enters the tympanum through the Glaserian fissure, supplying the mucous membrane of the anterior wall and drum membrane and anastomosing with the tympanic branches of the internal carotid

artery. (4) The *stylomastoid artery* (branch of the posterior auricular) anastomoses within the facial canal with the petrosal branch of the middle meningeal artery. It gives off several tympanic branches which enter the tympanum through small orifices in its posterior wall. It also sends a branch to the canal which terminates in the eminentia pyramidalis, supplying the stapedius muscle (Zuckerkandl).

From the Internal Carotid Artery.—The tympanic branch of the internal carotid enters the tympanum through an aperture in the carotid canal and anastomoses with the tympanic branches of the internal maxillary and stylomastoid arteries.

Veins.—The veins which remove the blood from the tympanic cavity correspond somewhat to the tympanic arteries. They leave the tympanum by various routes,—*i.e.*, (a) through the petrosquamosal suture, or minute foramina remaining after its obliteration, emptying into the veins of the dura mater; (b) through small foramina in the carotid canal, emptying into the carotid plexus; (c) by way of the Glaserian fissure, joining the veins about the temporomaxillary joint. There are also a large number of small veins which perforate the drum membrane at its periphery to join those of the external auditory meatus. Engorgement of these latter is the cause of the characteristic peripheral redness of the drum membrane, so often seen in tubotympanic congestion, or when the venous return flow has been impeded by continuous pressure upon the walls of the meatus, as by a speculum too large or retained in position too long.

Nerves.—The nervous supply of the middle ear is derived chiefly from the *glossopharyngeal*, the *sympathetic*, and the *trifacial*. The tympanic branch of the glossopharyngeal (Jacobson's nerve) is derived from its petrous ganglion, which is situated on the inferior part of the petrous portion of the temporal bone. It enters the tympanum through a small foramen in the tympanic floor, and, passing upward upon the promontory, divides into branches which are distributed to the round and oval windows and to the mucous membrane of the middle ear and the Eustachian tube. It also gives off branches of communication which lie in grooves upon the promontory and pass in different directions to their anastomotic connections. This division of the tympanic (Jacobson's) nerve into branches of distribution, together with its association with other nerves, constitutes the *tympanic plexus*. Its branches of communication are three in number. One of these passes downward and forward to unite with the tympanic branches of the sympathetic. A second communicating branch passes upward and forward to join the large superficial petrosal nerve. A third, receiving a small branch from the otic ganglion, passes upward in the substance of the petrous bone as the small superficial petrosal nerve. It receives in passing a filament from the ganglionic enlargement of the facial nerve.

The Sympathetic.—The tympanic branches of the sympathetic (nervi carotici-tympanici) are derived from the carotid plexus. They enter the

tympanum through small openings in the carotid canal, and there unite with filaments of the communicating branches of Jacobson's nerve, aiding in the formation of the tympanic plexus.

The Trifacial.—This nerve contributes to the nerve supply of the middle ear chiefly through the otic ganglion, which supplies two small branches, one to the membrano-cartilaginous Eustachian tube and the other to the tensor tympani muscle.

The stapedius muscle is supplied by a branch of the facial, the tensor tympani by a small branch from the otic ganglion.

The chorda tympani nerve traverses the tympanum without supplying it. It leaves the facial canal a little above the stylomastoid foramen, and passes obliquely upward and forward through a canal in the premastoid plate to enter the tympanum. It passes forward through the upper part of the atrium,—between the hammer handle and the long arm of the incus,—and leaves the tympanum at the Glaserian fissure through a canal known as the iter chordæ anterius. In its passage across the tympanum it is covered by the fold of mucous membrane which enters into the formation of the posterior pocket of the drum membrane.

The Eustachian Canal.—The Eustachian tube connects the cavity of the tympanum with that of the nasopharynx, and forms a very important part of the sound-conducting apparatus. Structurally it is somewhat analogous to the external auditory meatus in that it consists of two parts,—(1) an *osseous* and (2) a *membrano-cartilaginous* portion. Its length in the adult varies from 31 to 38 mm. (about 1¼ to 1½ inches), of which about one-third is bony and two-thirds fibrocartilaginous. The two portions of the tube are not quite in the same straight line, their point of junction forming an obtuse angle, opening downward. The general direction of the canal from the tympanum to the nasopharynx is forward, inward, and downward,—so that its pharyngeal orifice is anterior to, and on a lower level than, the tympanic orifice. The tube is somewhat expanded at either end, the vertical diameter of the tympanic orifice being from 3 to 4 mm., while that of the pharyngeal orifice is 5 to 8 mm. The narrowest part of the tube is where the bony and cartilaginous portions join (the isthmus), at which point the calibre of the bony tube is not more than 1½ to 2 mm. There are, however, parts of the membrano-cartilaginous tube in which the calibre is made even less than this by approximation of the anterior and posterior walls.

The osseous portion of the Eustachian tube extends from the anterior wall of the tympanum forward, inward, and downward through the upper and outer portion of the os petrosum. It is in immediate relation above with the canal for the tensor tympani muscle, this being roofed over by a continuation of the tegmen tympani. Internally it is separated by a thin plate of bone from the canal for the internal carotid artery. From its tympanic orifice the calibre of the tube becomes gradually smaller, reaching its minimum diameter at its anterior, or inner, extremity, which

presents somewhat roughened edges for the attachment of the cartilaginous portion of the canal.

THE MEMBRANO-CARTILAGINOUS PORTION OF THE EUSTACHIAN TUBE is attached to the base of the skull, and at the isthmus rests in the groove between the inferior border of the greater wing of the sphenoid and the petrous portion of the temporal bone. It commences at the anterior inner end of the bony canal, and from this point extends forward, inward, and downward to its orifice in the lateral wall of the nasopharynx. Its cartilaginous frame is not complete,—only its posterior wall, roof, and upper part of the anterior wall being cartilaginous, the remainder being completed by fibrous tissue (Fig. 30). Its posterior (median) wall is formed by a plate of fibrocartilage which becomes wider as it extends forward and inward, its pharyngeal margin causing a somewhat vertical projection upon the lateral wall of the nasopharynx. This projection, or

FIG. 30.—Transverse section of Eustachian tube (after Schwalbe, reduced).

ridge, forms the posterior wall of the pharyngeal mouth of the tube, and the anterior wall of the fossa of Rosenmüller. The upper margin of the cartilage of the posterior wall curls forward and then downward, being thus continuous with the roof and cartilaginous plate of the anterior wall. The narrow groove between the anterior and posterior plates forms the roof of the canal. The anterior (external) cartilaginous plate is narrow and forms only one-fifth of the anterior wall of the tube. Below this the anterior wall and floor are formed by a dense fibrous membrane (tunica propria) which is richly supplied with blood-vessels. The fibrous layer is continuous with the perichondrial lining of the cartilaginous portion of the tube (Rudinger).

The mucous membrane lining the Eustachian tube is of the ciliated cylindrical variety. In the pharyngeal end of the canal it is thicker and more loosely applied, and upon the anterior (membranous) wall it forms several longitudinal folds which admit of a certain degree of expansion of the tube without stretching the mucosa.

Much has been written, and many varying views advanced, as to the

patency of the Eustachian tube when the palatal muscles are at rest. Since post-mortem conditions, as shown by cross-sections of the tube, can not be relied upon as determining the condition of the parts in the living subject, it seems safest to confine one's statements to demonstrable facts. In all parts of the membrano-cartilaginous tube its vertical diameter far exceeds the lateral, which in most situations is obliterated by the contact of opposing surfaces. Owing to the absence of a complete cartilaginous frame, the anterior (membranous) wall is in contact with the posterior wall. In the upper part of the tube, however, the narrow cartilaginous plate which forms part of the anterior wall should tend to preserve the patency of this part of its lumen. It is this upper portion of the lumen of the membrano-cartilaginous tube which represents the direct continuation of the bony canal. It is probable that in health the lumen of the osseous canal is always patent; and that its anterior extremity, acting as a support, aids in maintaining the patency of the contiguous portion of the membrano-cartilaginous tube. The patency of the pharyngeal end of the tube is also, in all probability, maintained by its better cartilaginous support in this situation. Between these two terminal portions of the fibrocartilaginous tube, it seems probable that its anterior and posterior walls are for the most part in contact except when acted upon by the palatal muscles.

Anatomical Differences Between the Eustachian Tubes of the Adult and the Infant at Term.—In the new-born infant the Eustachian canal presents the following marked variations from the adult type: (1) It is very much shorter, measuring not more than 14 or 15 mm.[2] (33 to 38 mm. in the adult). (2) The tympanic orifice and the calibre of the bony tube are quite as large as in the adult. The whole canal is, therefore, in proportion to its length much wider. (3) The two portions of the tube (*i.e.*, the membranous and the bony) are more nearly in the same straight line, so that there is no demonstrable angle at their point of junction. (4) The whole tube is nearly horizontal in direction, so that, while the pharyngeal orifice in the adult is on a lower level by 12 to 14 mm. than the tympanic orifice,[3] it is on the same plane as the latter in the infant at term. (5) The pharyngeal mouth of the tube in the infant at term is on the same level as the hard palate,—*i.e.*, just behind the choanæ,—whereas in the adult it is not less than 10 mm. above the hard palate.

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[2] The statement found in many text-books, that the Eustachian canal of the infant at birth is 18 to 20 mm. long, is not correct. Remembering that the infant tube is nearly or quite horizontal in direction, examination of the base of the skull of an infant at term seems to demonstrate clearly that the length of the entire tube can not at this period exceed 14 or at most 15 mm.

[3] The statement of Politzer (Diseases of the Ear, p. 38), repeated by Brühl (Atlas of Otology, p. 37), that the tympanic orifice is in the adult on a level about 2.5 cm. higher than the pharyngeal orifice, is obviously incorrect, since, with Politzer's estimated length of 34 to 36 mm., this difference in the level of its two extremities would bring the direction of the canal into a plane much nearer the vertical than it is known to occupy.

The above anatomical differences, while undergoing fairly rapid modification with the growth of the child, may be accepted as characteristic of the tubal type in infancy as compared with the adult type.

Muscles of the Eustachian Tube.—When at rest, the Eustachian canal is supposed to be practically a closed tube. At frequent intervals, however, it is made to dilate in response to the action of two muscles, which must, therefore, be briefly described.

The *levator palati* arises from the anterior part of the under surface of the petrous bone, and passes obliquely downward and inward along the outer wall of the nasopharynx to be attached to the posterior surface of the soft palate. In its course toward its palatal attachment, some of its fibres are attached to the floor of the membranous canal. Its contraction serves to elevate the soft palate and to dilate the Eustachian tube by elevating its floor. The levator palati is supplied by the great superficial petrosal nerve (from the superior maxillary branch of the 5th).

The *tensor palati* muscle consists of two portions, which are sometimes spoken of as the vertical and horizontal portions respectively. The vertical portion arises from the scaphoid fossa at the base of the pterygoid plate, from the spine of the sphenoid, *and from the anterior membranous wall of the Eustachian canal.* These fibres unite to form a broad, thin muscle, which passes downward and slightly forward to the hamular process of the internal pterygoid plate. The muscle then winds around the hamular process and passes inward (horizontal portion), to be attached to the anterior surface of the soft palate. Its contraction elevates the soft palate and dilates the Eustachian tube by drawing its anterior wall downward and forward and thus away from the posterior wall. This muscle is supplied by the otic ganglion.

There is still another small muscle, the *salpingo-pharyngeus*, the contraction of which influences the calibre of the tube. It arises from the tubal prominence, or pharyngeal end of the posterior cartilaginous plate of the tube, and passes backward to be inserted into the posterior pharyngeal wall.

The Mastoid Process (Plate III).—The mastoid process is the large conical protuberance of bone which projects downward behind and below the bony meatus. It is bounded above by the tegmen antri, which separates it from the middle cerebral fossa. The interior of the upper part of the mastoid process communicates with the tympanic vault, which lies immediately in front. Below this it is separated by the dense bony plate of the posterior canal wall from the osseous meatus. Inferiorly it projects downward below the level of the floor of the meatus, terminating in the conical process known as the tip (Fig. 31, *b*). The outer surface is convex (31, *a*) and more or less roughened for the attachment of muscles. The inner surface of that portion of the mastoid lying above the floor of the meatus forms part of the posterior cerebral fossa. It presents the deep groove (32, *a*) which lodges the descending portion of the lateral sinus.

The position of this groove in its relation to the tympanum varies greatly in different skulls. It is one of the most important surgical relations of the tympanum, and will be considered more fully in a later chapter dealing with the surgical anatomy of the temporal bone. The inner surface of the tip—that portion of the mastoid which projects downward below the base of the skull—forms the outer boundary of a groove, running from before backward, for the attachment of the posterior belly of the digastric muscle (31, *c*).

The mastoid process consists of an outer shell, or cortex, of dense compact bone, inclosing a central chamber, which may be subdivided into pneumatic spaces or filled with diploic tissue. Accordingly two types are recognized, viz.:

(1) The PNEUMATIC (Fig. 34), in which the interior is subdivided into distinct spaces, which are lined with mucous membrane, and may be of very considerable size. In this variety there is invariably a large cell at the tip, in which pus is apt to collect during the course of a suppurative mastoiditis.

(2) The DIPLOIC MASTOID (Fig. 35).—In this variety, instead of distinct, pneumatic spaces, we find the interior of the mastoid filled with cancellous tissue, somewhat resembling that which occupies the space between the inner and outer plates of the cranial bones. In the dried bone this tissue appears as if composed of very small, thin-walled osseous cells. In the living subject, as seen during an operation upon the mastoid, it consists of a vascular, spongy tissue offering only moderate resistance to the rongeur or curette. Even in bones of this type there is usually present a single large cell at the tip. Between the two varieties above described, there are many bones which present the characteristics of each,—*i.e.*, pneumatic spaces and diploic tissue occupying different parts of the same bone.

Still another type of bone has been described as the SCLEROTIC MASTOID. In this variety there may be throughout the greater part of the mastoid process complete absence both of pneumatic spaces and of diploic tissue, their place being occupied by fairly compact bone. This solidification sometimes occurs as a pathological change resulting from chronic middle-ear suppuration of long standing. There are undoubtedly cases, however, in which the mastoid is practically filled with a solid mass, in spite of the fact that the tympanum may present no evidences of having been the seat of disease. The accompanying illustration (Fig. 36) was made from a specimen prepared by the author. That there had been no prolonged suppurative process within the tympanum was shown by the condition of the ossicles and drum membrane, which were intact and normal.

THE ANTRUM.—There is one space, usually of considerable size, which is always present,—viz., the so-called mastoid antrum. This is the large space situated in the upper and anterior part of the mastoid process immediately behind the tympanic vault. There can be no doubt that this space forms part of the tympanic cavity. Its roof is directly continuous with

PLATE III.

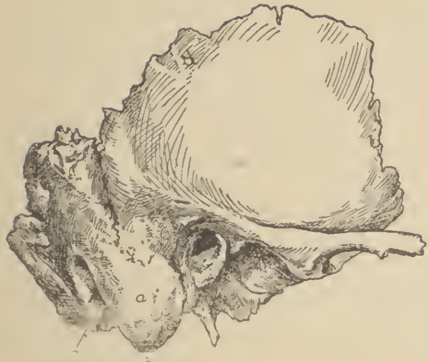

FIG. 31.—Temporal bone, outer surface; *a*, mastoid process; *b*, tip of mastoid; *c*, groove for posterior belly of digastric muscle

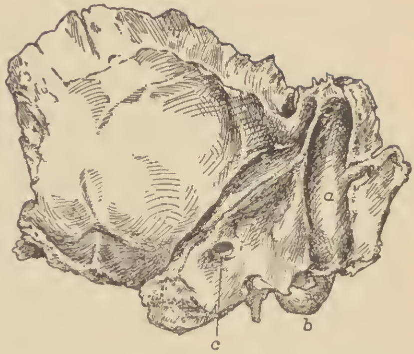

FIG 32—Temporal bone, inner surface; *a*, groove for sigmoid sinus, *i.e.*, descending part of lateral; *b*, tip of mastoid; *c*, internal auditory meatus.

FIG. 33.—Vertical section of temporal bone passing through aditus ad antrum; *a*, aditus, leading forward into tympanic vault.

FIG. 34.—Section through mastoid process of pneumatic variety, showing typical pneumatic spaces or cells.

FIG. 35.—Section through mastoid process of diploic type; *a*, antrum.

FIG. 36.—Section through mastoid process of sclerotic variety.

that of the vault, from which it is differentiated only by the triangular constriction known as the aditus ad antrum (33, *a*). The antrum is fully developed in the new-born,—*i.e.*, before even a rudimentary mastoid can be demonstrated. Later it is surrounded and appropriated by the developing mastoid process.

In this brief description of the mastoid process, the author has deemed it best to deal only with those general characteristics a knowledge of which is absolutely essential to the intelligent study and appreciation of the commoner acute middle-ear lesions. He has endeavored to emphasize the fact that the antrum is not to be considered separately as one of the mastoid cells, but rather as the posterior end of the tympanic vault. This method of describing it enables the student more easily to remember its exact position and its surgical significance. It has the further advantage of being, from the stand-point of its development, the only correct view; for even in fetal life—*i.e.*, long before even a rudimentary mastoid is present—the antrum is an easily demonstrable cavity.

Physiology of Sound-Conduction.

It is impossible to consider the process of sound-conduction without having in mind both the physics of sound and the physics and physiology of the conducting mechanism. With special students of otology, more or less familiarity with the physics of sound may be assumed. For the busy medical practitioner, however, it may not be amiss to recall briefly certain elementary, though perhaps forgotten, facts upon which our knowledge of the physiology of sound-conduction is based.

Sound is a form of motion produced by some vibrating body. A sound-wave is a series or chain of alternate condensations and rarefactions in the surrounding air, by which the vibratory movements of the sounding body are conveyed to the drum membrane. When these vibrations occur singly or follow each other irregularly, the resulting sound is discordant and is spoken of as a noise. When the vibrations recur rhythmically or at regular intervals, and with sufficient rapidity, a musical tone results.

The *pitch*, or relative position of sound in the musical scale, depends upon the rapidity with which the vibrations follow one another, the pitch being raised as the number of vibrations per second is increased. The human ear normally appreciates musical tones between two extremes of vibration rapidity, the lower tone limit being eighteen vibrations per second, and the upper tone limit somewhere between 32,500 and 50,000 vibrations per second. Vibrations recurring rhythmically at a rate of less than eighteen per second may be heard as separate impulses, but are not heard as a continuous musical tone. Taking any note in the musical scale,—*e.g.*, C^{-2}, 32 double vibrations,—by doubling the number of vibrations we may produce a tone one octave higher. Thus 32, 64, 128, and 256 double vibrations per second represent C tones one octave apart. Pitch, then, is determined solely by the number of vibrations per second.

Intensity.—The intensity with which a sound-wave impresses the perceptive mechanism depends upon its amplitude of vibration. Taking for example a tuning-fork such as is represented by Fig. 37, a double vibration includes the passage of the prong from *a* to *b*, its recoil to *c*, and its return to *a*, its position of rest. The line *b-c* represents what is called the amplitude of vibration. Supposing this to represent its vibration when set in motion by a moderate tap, a more vigorous blow will result in more extensive movements, the amplitude of vibration being greater and the intensity of the sound being correspondingly increased. This is in accordance with the physical law which declares that *the intensity of sound is proportional to the square of the amplitude.*

Fig. 37.—Tuning-fork.

Timbre.—There is a third property of musical sound, that peculiar quality by which we are able to distinguish tones of the same pitch as produced by different musical instruments; *e.g.*, the C tone of the piano, of the violin, and of the human voice. For an explanation of this quality of sound, and of the harmonics or overtones to which it is due, the reader is referred to any of the standard works on physics.

Sound-Conduction.—*The Auricle and External Auditory Meatus.*—To most of the lower animals the auricle is undoubtedly of great value in aiding them to concentrate their auditory effort at will upon some particular sound by turning the auricles in the direction from which it comes. In man, on the other hand, the auricle has lost much of its importance as a part of the conducting apparatus since the auricular muscles have lost the property of voluntary contraction,—in other words, since man has lost the power of moving the auricles at will. That he still retains an intuitive appreciation of their function is shown by the impulse which leads the hard-of-hearing instinctively to draw the ear forward toward the source of sound and to augment its concavity by that of the hollow of the hand. While it is now recognized that fairly good hearing is not inconsistent with complete loss of the external ear, it can not from this be assumed that the auricle is without influence upon the auditory function. Politzer has called attention to the fact that by filling the concha with wax, even though the orifice of the external auditory canal be left open, we reduce considerably the acuteness of audition; whereas obliteration of the other auricular depressions—*e.g.*, the fossa of the helix, fossa of the antihelix, etc.—does not influence the hearing appreciably. The concha, therefore, must be regarded as in man the most important part of the auricle, its

function being to receive and carry forward the sound-waves to the inner surface of the tragus, by which they are deflected into the external auditory meatus. The *external auditory canal* presents curves and variations in calibre in different parts, which undoubtedly have to do with the proper focusing of sound-waves upon the membrana tympani. These anatomical pecularities, however,—provided that its lumen is not completely occluded,—seem to be less essential to perfect hearing than almost any part of the conducting apparatus. Individuals with abnormally narrow auditory canals seem often to hear equally as well as others with canals of normal calibre. The author has seen several cases in which the bony meatus was very considerably narrowed in one or more of its diameters by exostoses from one or other of its walls, in which no appreciable impairment of hearing could be demonstrated. Another phenomenon with which every aurist is familiar is the excellent hearing power frequently exhibited by individuals whose auditory canals are apparently filled with cerumen, exfoliated epithelium, or other foreign substances. Whether in such cases the obstructing mass vibrates in unison with the sound-waves from without and thus transmits them to the column of air between it and the drum membrane, or the sound-waves are projected through some space between the foreign body and one of the canal walls, may be a debatable question. The latter hypothesis seems the more plausible. Sound-waves, then, are collected in the concha, whence they are deflected by the inner surface of the tragus into the external auditory canal, by which in turn they are conveyed to the outer surface of the membrana tympani.

The Drum Membrane.—In the membrana tensa we have an ideal medium for the transmission of sound-waves to the ossicular chain. It is a tense, inelastic, fibrous membrane, drawn inward at the centre by its attachment to the lower half and extremity of the hammer handle, and presenting, therefore, a somewhat funnel-shaped concavity toward the meatus. From the central depression at the umbo to its peripheral attachment, the membrane is slightly bowed or curved in the direction of the meatus. This outward curvature of the normal membrane is not without influence upon its value as part of the conduction mechanism, Helmholtz having established by experiments the greater resonance of curved membranes as compared with flat ones.

If the drum membrane were highly elastic it would respond easily to sound-waves, but would hold these and other vibrations due to harmonics, or overtones, after the original stimulus had spent itself; and these after-tones would interfere with the proper transmission of sound-waves immediately following. But the membrana tensa is a comparatively unyielding and inelastic structure, responding readily to all forms of sonorous stimuli, but returning rapidly to a condition of rest as soon as it ceases to be acted upon by sound-waves from without. As with all stretched membranes and sonorous bodies generally, the membrana tensa has a fundamental

note of its own. By this is meant that the drum membrane, if set in motion by a light tap, or any force momentarily bending it, must execute a series of to-and-fro movements, or vibrations, before it can return to a condition of rest; and that these movements will be rhythmic in character and will take the form of sonorous vibrations having a constant rate of rapidity per second; in other words, they would if sufficiently extensive and prolonged produce a musical tone of constant pitch, its *fundamental tone.* One disadvantage of a loud fundamental note in the drum membrane would be the confusion which would inevitably result if it were acted upon by sound-waves having the same number of vibrations per second. It must be remembered that the drum membrane is capable not only of responding to all kinds of sonorous impressions, but of transmitting several sound-waves simultaneously. One can readily appreciate, therefore, that a fundamental tone of any part of the conducting mechanism which would respond loudly even to overtones of the same pitch might cause great disturbance of function. Against this result, the handle of the malleus acts as a control mechanism, the effect of its attachment to the drum membrane being somewhat analogous to the pressure of a finger against the prong of a vibrating tuning-fork,—*i.e.*, the vibrations due to the fundamental tone of the drum membrane are immediately overcome by the inertia of the attached ossicular chain, leaving the membrana tensa free to receive and transmit new sound-waves from without.

The Ossicular Chain.—According to the commonly accepted theory, sound-waves striking upon the membrana tympani are by it communicated to the ossicular chain, by which in turn they are transmitted through the foot-plate of the stapes to the labyrinthine fluids. Each excursion in either direction of the stapes is accompanied by a corresponding movement, or displacement, of the fluid in the vestibule, and in this way fluid waves are inaugurated which finally impress the terminal filaments of the auditory nerve. For the exercise of these passive movements, the drum membrane and ossicles constitute a perfectly adapted mechanism. The ossicles form a system of finely adjusted and sensitive levers which respond instantly to the slightest movements of the drum membrane. The handle of the malleus being the only part of the ossicular chain directly attached to the membrana tympani, it is through this process that the movements of the drum-head are transmitted to the ossicles. The handle of the malleus is longer than the long arm of the incus; so that, according to the laws governing leverage, with each inward excursion of the hammer handle, the long arm of the incus moves inward, carrying with it the stapes through a shorter distance but with greater force. This provision has a particularly important bearing upon the transmission of the lower musical tones, which are produced by vibrations of large amplitude but relatively little force. The funnel-shaped concavity of the drum membrane and its outward curve from centre to periphery have also an important bearing upon the

function of the conducting mechanism,—such a membrane, according to Helmholtz, transmitting low vibrations of smaller force than would a flat membrane. Apparently, therefore, the ossicular chain is most essential in transmitting the lower tones of the musical scale,—tones produced by vibrations of large amplitude but little force, which the ossicular levers convert into vibrations of smaller amplitude but of greater force. The higher tones, on the other hand, are produced by sonorous vibrations of smaller amplitude, but whose relatively greater force is capable of inaugurating a fluid wave within the labyrinth without augmentation of force through the leverage system of the ossicular chain. With this theorem in mind, we should be at no loss to explain the impairment of hearing for the lower tones of the musical scale which invariably attends any severe lesion of the conducting mechanism.

The relative movements of the different ossicles have been investigated by Helmholtz, Politzer, and others. According to their experiments, the maximum excursion of the hammer handle (at umbo) which can be induced by condensation and rarefaction of the air in the external auditory canal is ¾ mm., the maximum movement of the stapes being 1/14 to 1/18 mm. These measurements throw light on the influence of leverage on the ossicular movements, and furnish additional proof that the ossicles move separately and individually as parts of a movable chain, and never in unison as parts of a rigidly bound whole. It must be understood, however, that the measurements given represent only the excursions which may be experimentally induced, Riemann having proved by mathematical calculations that the largest excursion of the stapes possible in response to sound-waves would be smaller than the eye could appreciate.

Another view, which has been entertained, as to a possible pathway for sound-waves is that they may reach the labyrinth by way of the round window. Thus, Politzer [4] states that "the vibrations of the drum membrane are transmitted to the labyrinth partly through the ossicles by means of the foot-plate of the stapes, and partly through the air in the tympanic cavity to the membrane of the fenestra rotunda." The writer can not conceive how in a healthy ear it is possible for the membrane of the round window to take so direct a part in the transmission of sound-waves. To accept such a view, it would be necessary to assume that, with each inward movement of the membrana tympani, the membrane of the round window is also forced inward by condensation or displacement of the air in the tympanic cavity, an untenable hypothesis. The membrane of the round window moves inward toward the lumen of the scala tympani only during the period of rarefaction of sound-waves,—*i.e.*, during the period in which the stapes moves outward. In other words, its function seems purely one of compensation for the movements of the stapes, thus preserving the stability of intralabyrinthine pressure.

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[4] Diseases of the Ear, last English edition, p. 57.

The Tympanic Muscles.—The function of the tensor tympani and stapedius muscles has been the subject of a great deal of controversy. Contraction of the tensor tympani, drawing the hammer handle and drum membrane inward, renders the latter more tense. Contraction of the stapedius draws the head of the stapes backward and the anterior end of the foot-plate of the stapes somewhat outward into the niche of the oval window. Acting together, the two muscles oppose each other, one drawing the ossicles inward, the other tending to draw the stapes outward. In this way it is believed that they may constitute a sort of control mechanism, guarding the labyrinth, on the one hand, from the effect of loud or explosive noises which might drive the stapes violently inward, and, on the other, neutralizing the effect of sudden and forcible condensation of the air within the tympanum which might cause excessive excursions of the hammer handle and drum membrane in the other direction.

Another theory is that which was proposed by Mach as long ago as 1863. According to this view, the muscles act as an accommodation apparatus, varying and regulating the tension of the conducting mechanism according to the requirements of different sound-waves, and also enabling the individual by an effort of will to select and follow certain sounds. This view, with some modifications, has received the support of most later observers. Henson, in 1876, showed by experiments upon living animals that the tensor tympani muscle not only contracts in response to sound-waves, but executes a distinct contraction for each particular sound; and further that its contractions were stronger for high tones than for the lower tones of the musical scale. Giving these facts greater significance were the later experiments of Pollak, which proved that after removal or destruction of the cochlea in living animals the tensor tympani no longer contracts except in response to electrical stimulation. While absolute proof as to their function is beyond our reach, this view—viz., that different sound-waves require variations in the tension of the sound-conducting mechanism for their perfect transmission, and that such variations are controlled and regulated by the tympanic muscles—is in harmony with laboratory investigations, and furnishes the only hypothesis which seems adequately to explain these perfect little muscles.

A question which is still the subject of occasional discussion, and apparently with little prospect of definite solution, is whether sound-waves are in any degree conducted through the ossicles and labyrinthine fluids by molecular motion, or altogether through vibration of these structures en masse. The fact that the higher musical tones are in many cases well perceived in spite of advanced obstructive lesions in the conductive mechanism, is by some regarded as pointing to a possible transmission of these sounds by molecular processes, rather than by mass movements of the ossicular chain. In this regard the following expression of belief by the

late Professor Bezold[5] of Munich is of interest: "I share the conviction . . . that not molecular movements, but mass movements of the conducting chain, together with the labyrinthine water column, transmit the sound-waves from the air; . . . I am, moreover, on the side of those physiologists who regard bone conduction as osteotympanic. In other words, in conduction through bone, the excitation of the auditory nerve takes place exclusively with the aid of the sound-conducting apparatus, vibrating with the bone." This view, while not final, probably expresses the belief of a majority of otologists and physiologists to-day.

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[5] Archives of Otology, vol. xxxv, No. 3, p. 217.

CHAPTER II.

PHYSICAL EXAMINATION OF THE PATIENT.

UNDER this heading should be included a short but comprehensive history of the case; and this should include not only all essential data as to the present attack, but also an account of any previous conditions or disorders which may throw light on the aural disease.

Age, Occupation, Habit, Heredity.—Age has a particularly important bearing upon aural disease and should always be taken into account. The patient's occupation and habit or mode of life—*e.g.*, environment, excessive addiction to alcohol or tobacco, habitual overwork, dissipation, etc.—should be kept in mind as having a possible bearing upon the aural affection. The question of heredity is apt to be considered chiefly in connection with adult patients suffering from impaired hearing. It might be taken into far more useful account in our dealing with the children of the deaf or hard-of-hearing, with whom especial effort should be made to correct any conditions predisposing to aural disease.

Previous History.—This should include a record of (A) ANY CONSTITUTIONAL DISEASE, inherited or acquired,—*e.g.*, syphilis or tuberculosis. (B) DIATHESES, of which gout and rheumatism are the most important. That rheumatism is the indirect cause of many cases of etiologically obscure aural disease there can be no doubt; and there are some observers (Buck, O. Wolf, Von Urcherman) who believe that acute rheumatism is not infrequently manifested as a primary lesion within the tympanum. (C) THE ACUTE INFECTIOUS DISEASES, either in childhood or in later life. At least 8 per cent. of all cases of scarlet fever, diphtheria, and measles develop some form of middle-ear inflammation. Particularly are many cases of chronic suppurative otitis media traceable to such lesions early in life. (D) PREVIOUS ATTACKS OF ACUTE OTITIS MEDIA. There are many children whose histories are completely negative as to infectious diseases who have, however, experienced two or more attacks of acute tympanic inflammation. Recurring attacks of aural pain and discharge, for which no exciting cause can be assigned, should always suggest the possibility of some abnormal condition within the nasopharynx. Certainly such a history should make one more guarded in one's prognosis until the nasopharyngeal lesion, whatever it may be, has been corrected.

History of the Present Attack.—This should begin with a statement of the disease to which the aural disorder was secondary, or with the exciting cause. In order to fix as nearly as possible the date of the onset, we should begin with the initial symptom—pain, deafness, tinnitus—and the date on which it was first experienced by the patient. In acute cases, next to the initial symptom, it is important to learn if possible the time which has elapsed between the onset of the attack and the appearance of

the aural discharge, provided this be present. The importance of this lies in the fact that in acute tympanic disease the prognosis is, as a rule, more favorable in those cases in which spontaneous rupture of the drum membrane follows quickly upon the initial symptom than in cases in which it is delayed several days or a week. Further than this but little can usually be learned beyond what the physician's examination will bring to light,—unless, of course, the patient has been under the care of a trained observer. It is, of course, well in the case of a seriously ill patient to inquire as to whether rigors, profuse sweating, or sudden and pronounced alterations of temperature have been noticed; but the statements of the family or attendants in regard to such symptoms are rarely to be relied upon. In other words, the physician is usually obliged to await the results of his own observation on these points.

In cases of chronic aural disease the history may extend over a period of months or as many as ten or twenty years. Obviously such cases require a more thorough weighing of individual symptoms, their sequence, and relative severity or prominence in different stages of the disease.

To epitomize: The main points of interest in the history of a patient suffering from tympanic disease group themselves about the following facts:

(1) The primary disease to which the otitis was secondary, or the exciting cause.

(2) The date and character of the onset.

(3) In acute cases, the date and character of the discharge.

(4) The presence or absence of symptoms of constitutional disturbance,—nausea, vomiting, rigors, septic temperature, etc.

(5) In chronic cases, the sequence of the various symptoms, and their relative severity in different stages of the disease.

(6) The course of the disease as shown by subsidence or persistence, increase or diminution of symptoms.

From what has been written, it may seem that the writer has proposed a somewhat voluminous history. This, however, is not necessary, it being quite possible to include all that is of practical importance within a comparatively small space. To facilitate this, some form of printed history blank is almost essential. The writer has found two forms of history cards useful,—*i.e.*, one for office histories, used mostly in cases of chronic aural disease (*e.g.*, deafness, etc.), and a more abridged form for "bedside histories," which are of great convenience in cases of acute tympanic or mastoid disease. Copies of these two forms will be found in a final chapter or appendix.

Objective Examination.—Before attempting to inspect the drum membrane by reflected light, much may be learned by a careful observance of the patient. One may often determine at a glance whether the patient breathes normally—*i.e.*, through the nose—or is a typical or partial mouth-breather. Habitual mouth-breathing in children is usually due to the presence of adenoids. This obstacle to normal breathing is, however,

by no means invariably announced by the typically dull and vacant expression so often described in the earlier text-books. The child is often of extremely healthy appearance. He may breathe quite normally during the day or when perfectly quiet, but with noticeable difficulty at night when the recumbent position favors nasopharyngeal congestion, or when engaged in any active physical exercise. While, therefore, we may often determine the presence of adenoids at a glance, our ability to do so can not be relied upon. Mouth-breathing in adults is less frequent, and usually, therefore, more noticeable than in children. Whatever the patient's age, habitual mouth-breathing should be noted as likely to throw light not only upon the etiology, but also upon the prognosis and treatment of the aural disease.

In talking to a patient with advanced catarrhal deafness, one intuitively gauges the degree of deafness by one's difficulty in making him hear or by the evident difficulty on his part; also which is the deafer ear, by observing which ear is turned toward the speaker. By observing the patient's expression and the intentness with which he scans the face of the person talking, one not infrequently discovers a capacity for lip-reading of which the patient himself may be only partly conscious. By such practical observations one may obtain very useful information as to the degree of deafness for which relief is sought.

Auricular Displacement.—Of great importance, because of their possible surgical significance, are changes in the position of the auricle due to inflammatory processes in or about the ear. Note if the two auricles are identical in the angles which they form with the sides of head. While slight differences in this respect may represent simply an anatomical variation, marked differences are much more likely to be the result of an inflammatory condition in the ear or temporal bone of one or the other side. Marked projection outward and forward, or outward and downward, of one auricle beyond the position occupied by the opposite ear, points usually to an inflammatory process either in the mastoid cells (acute mastoiditis) or in the posterior wall of the fibrocartilaginous meatus (furunculosis). This outward displacement of the auricle is due either to a subperiosteal abscess or to œdema of the structures behind the ear,—*i.e.*, those covering the mastoid process. The postauricular sulcus—linear depression between the auricle and side of the head—may therefore be obliterated. Such postauricular swelling, with outward displacement of the auricle, points in young children almost invariably to a suppurative inflammation involving the mastoid cells. In the adult, on the other hand, they may mean mastoid inflammation, with resulting subperiosteal abscess, *but far more frequently are the result of an extension of inflammation from a furuncle in the posterior wall of the fibrocartilaginous meatus.*

Palpation in Case of Postauricular Œdema.—With œdema behind the auricle much may be learned as to the original focus of infection by careful palpation. Gradually exerted, but firm, pressure at a point upon the mastoid process just behind the auricular attachment, but so

directed as not to disturb the auricle, will elicit deep-seated bone tenderness only if the mastoid process is the seat of an acute inflammatory process. If the mastoid is not involved, little or no pain will result from pressure so directed. On the other hand, pressure at exactly the same point, but directed slightly forward so as to move the auricle, will cause the patient to wince or cry with pain, even though the mastoid is perfectly healthy, if the fibrocartilaginous meatus be the seat of an acute inflammation. This latter phenomenon is, of course, explained by the fact that any movement of the auricle along the line of its attachment to the side of the head is necessarily communicated to the cartilaginous framework of the external auditory canal. It is a differential point of very considerable diagnostic importance, and will be referred to again in a later chapter.

Palpation of the Auricle in Case of Aural Pain.—Whenever ear pain is complained of, it is well to determine, if possible before any attempt is made to examine the drum membrane by reflected light, whether this pain is in any part due to an inflammation of the external canal. Except in infants and very young children, in whom the anatomical relation between the drum membrane and external meatus is exceedingly close, manipulation of the auricle causes absolutely no pain when the inflammation is confined to the middle ear. On the other hand, very slight movements of the auricle are extremely painful even in the initial stages of a furuncle in the meatus. By pressing one's finger firmly against the cheek immediately above and in front of the auricle, and bringing it downward along the anterior attachment so as to press the tragus inward, one will elicit tenderness in every case in which the anterior wall of the meatus is the seat of an acute inflammation. If no pain results from this procedure, the auricle should be moved in different directions,—upward, backward, downward, and forward. These movements will change the position of the different walls of the meatus, and if no pain is experienced one may with confidence exclude the fibrocartilaginous canal as the seat of an acute inflammation.

If we have properly cultivated our powers of observation, we have now with the expenditure of but a few moments of time possessed ourselves of certain facts bearing upon the patient's condition. We have made a practical estimate of the degree of functional impairment (deafness) by his difficulty in interpreting the conversational voice, have noted his capacity for lip-reading and the probable influence of this in supplementing his power of audition, and have probably determined which is the deafer ear. If his disease is acute, we have either located the inflammation in the auricle or external auditory canal, or have excluded that part of the conducting mechanism as without bearing upon the aural disorder.

The next step in the examination is the inspection of the drum membrane. For this purpose, the following mechanical aids are essential: (a) some form of artificial light, (b) a forehead mirror for focusing the light upon the membrana tympani, and (c) a properly constructed aural speculum. These appliances are found in the instrument houses in a variety

of designs. While some of them are excellent in every respect, others are so far from correct in design and structure as to be practically without value in aural work. It is important, therefore, that a brief statement be made as to the essential points which these instruments should possess.

The Forehead Mirror.—In selecting a mirror for otological work, the points to be looked to are its size, the size of its central orifice, the position of the ball by which it is attached to the head-band, and, most important of all, its focal distance. Within certain limits, the size is a matter of personal preference, some instrument dealers showing them in various sizes from 2¼ to 4 inches in diameter. In the writer's experience, a mirror 3 inches in diameter has represented the most satisfactory size. This

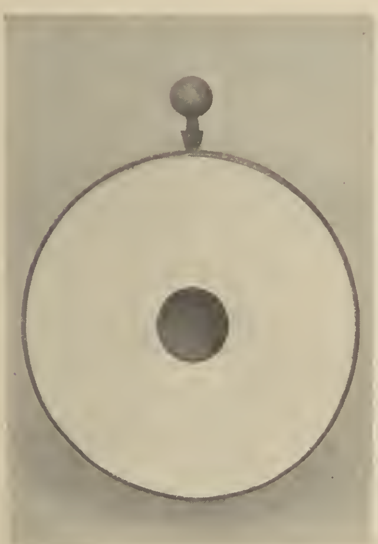

Fig. 38.—Head mirror.

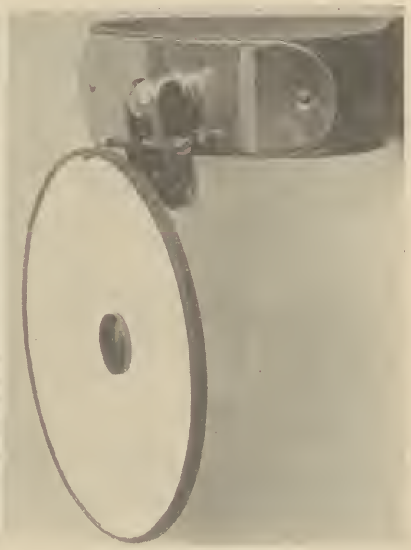

Fig. 39.—Head mirror.

throws a stronger light than those of smaller size, and, on the other hand, is lighter and less clumsy than larger ones. A 3½-inch mirror, however, if its attachment to the head-band is properly adjusted, can be used very satisfactorily and is the choice of many aurists. The central orifice should be not less than one-quarter of an inch in diameter, a smaller aperture entailing a certain amount of unnecessary eye-strain. The writer's personal preference is for a central orifice one-half inch in diameter, as shown in Fig. 38. This is particularly advantageous in tympanic surgery and in the post-operative treatment of mastoid wounds, a smaller aperture being apt to necessitate more frequent readjustments of the mirror, a serious inconvenience when one wishes to avoid touching anything not absolutely sterile. The ball by which the mirror is connected with the head-band should be attached to the peripheral edge (Fig. 38), rather than to its

posterior surface as shown by Fig. 39. The advantage of the peripheral attachment is that it allows a wider range of movement and also enables one to bring the central orifice nearer the eye.

Undoubtedly the most important point to be investigated in selecting a mirror for aural work is the focal distance. By this is meant the distance between the concave reflecting surface of the mirror and its principal focus, or that point in front of it at which the reflected rays converge to give the greatest brilliancy of illumination. It can be readily understood that a focal distance which would be quite satisfactory in examining the pharynx might be altogether unsuitable to the inspection of

Fig. 40.—Head-band and mirror.

so small a field as is afforded by the membrana tympani. Thus, a focal distance of 18 inches may afford a clear definition of the structures of the throat and pharynx, but would be altogether too great for the examination of structures so small as the minute landmarks of the drum membrane. For examining the ear, the focal distance should be not greater than 10 or 12 inches. This may be easily tested by holding the mirror with its reflecting surface about twelve inches from the flame of a candle, lamp, or gas jet, and noting the distance at which the outline of the flame is most clearly shown upon any flat surface,—*e.g.*, a book-cover or the palm of the hand. This is also the point at which the convergent rays meet to give the most brilliant illumination.

The Head-band.—Of this but little need be said beyond the mention of certain points to be avoided. A head-band of any elastic material is unsuitable, because it allows a degree of motion which is precisely what one wishes to avoid. The flexible band should be made, therefore, of leather or of very stout silk ribbon at least an inch wide. The attachment of the mirror to the forehead piece should be effected by means of a double ball-and-socket joint, as shown by Fig. 40. A single joint does not allow of placing the mirror in the position which gives the least eye-strain and the clearest vision. Fig. 41 represents a stiff fibre head-band which the writer has found very satisfactory in practical use. If properly fitted to the head, it is comfortable, and easily removed and put on. It has also the additional

Fig. 41.—Fibre head-band.

advantage over the ordinary head-band that it is more easily put on by an attendant or nurse during a surgical dressing, and usually falls automatically into its proper position on the head.

To epitomize: If one were obliged to depend upon ordering a mirror and head-band by mail, one would do well to include the following specifications: Diameter of mirror 3 to 3½ inches; diameter of central orifice not less than one-quarter of an inch; attachment ball to be on the peripheral edge of the mirror; focal distance 10 or 12 inches; head-band to be of stout unyielding material, and attachment mechanism to be provided with a double ball-and-socket joint. A mirror fulfilling these requirements should prove quite satisfactory in otological work.

Fig. 42.—Aural specula.

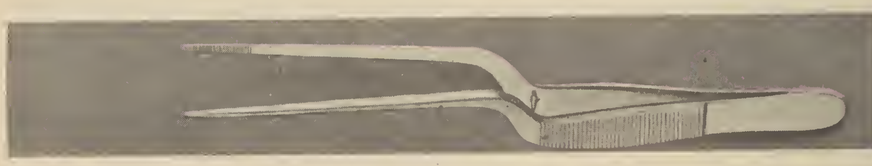

Fig. 43.—Aural forceps.

Fig. 44.—Tympanic probe.

Fig. 45.—Aural cotton applicator.

Aural Specula (Fig. 42).—The accompanying illustrations represent some of the specula which are offered for sale, and with any of which the ear may be satisfactorily inspected by a competent aurist. In making one's choice one should bear in mind the mechanical results which the speculum is intended to facilitate. What one needs is not a long and narrow funnel which can be introduced deeply into the external auditory canal, but rather a short tube for introduction into the orifice of the fibro-cartilaginous meatus. Any unnecessary length of the tubular end of the speculum is a hindrance to inspection and the facile use of instruments. The outer expanded portion of the speculum serves a double purpose,—viz. (1) that of a handle by which it is held in place and manipulated, and (2) that of a receptacle for collecting the light from one's mirror and throwing it into the auditory canal. Any superfluous length of this portion of the speculum simply adds to the difficulties of inspection and local treatment of the ear. As a teacher of practical otology, the author has been interested in obtaining whatever speculum would best aid the medical student in acquiring quickly and easily a certain degree of facility in examining the ear. He knows of no more practical or satisfactory speculum than that shown in Fig. 42, *B*, known as the Boucheron speculum. They are made in sets of four sizes.

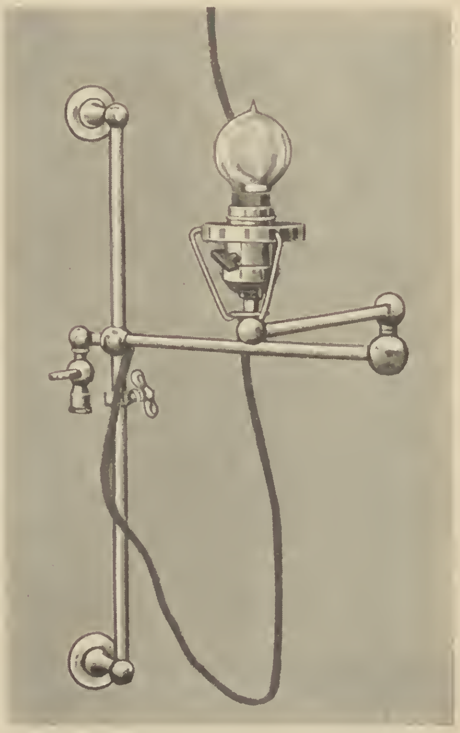

Fig. 46.—Wall bracket for movable electric light.

Three other instruments essential in aural examination are shown in the accompanying illustrations (Figs. 43, 44, and 45).

The Source of Light.—Excellent light for otological work can be obtained from the flame of an oil lamp or gas jet, or from an electric bulb connected with the ordinary street current. Whatever the source may be, the light itself must be connected with some form of movable bracket which can be moved both in the vertical and horizontal planes (Fig. 46). The electric light is now quite generally used in the cities, and, when properly installed, gives a satisfactory and suitable form of illumination.

Electric Forehead Lamp for Bedside Examinations.—Before leaving this subject, mention should be made of the lamp and pocket battery

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shown in Fig. 47. This little apparatus, which dispenses altogether with the mirror, has become an indispensable adjunct to one's equipment for bedside examinations. The battery is supplied with six dry cells, which, unfortunately, must be renewed every four or five weeks. This, however, is a trifling disadvantage compared with the difficulties of inspection by other methods of illumination in private houses.

Technic of Objective Examination; Landmarks of the Normal Drum Membrane.—Obviously our examination must be without value unless we have first prepared ourselves to recognize the physical characteristics of the normal ear. The student of otology will put his time to poor advantage unless his growth in technical skill and his knowledge of special anatomy are advanced simultaneously. The writer will, therefore, refer frequently to the anatomy of the parts under examination.

Fig. 47.—Forehead lamp for bedside examination.

As with all kinds of work requiring technical skill, each individual will inevitably in some degree develop his own method and technic. There are, however, certain points which the beginner will do well to bear in mind and adhere to strictly.

Position of Physician and Patient.—For aural examination it is much better that the physician should be seated to one side of the patient (Fig. 48), rather than directly in front of him with knees parted. This brings his eye within easy visual range of the ear to be examined and is the most comfortable position both for him and for his patient. To examine the opposite ear, it is easy to reverse this position either by revolving his own or the patient's chair, or by rising and assuming the same position on the other side of the patient.

Management of the Light.—The light should be about on the same horizontal plane as the patient's ear, and so placed that the rays from it to the mirror and from the mirror to the ear shall be as nearly as possible parallel; *i.e.*, they should form as acute an angle as possible. If the focal bistance of the mirror were exactly 10 inches, it would be better to bring doth the light and the ear under examination within that distance of the mirror. Usually, however, the focal distance is rather more than 12 inches, so that the best illumination is obtained by placing the light be-

FIG. 48.—Position of physician and patient for aural examination.

hind and to one side of the patient's head, making the distance between the light and the mirror at least 18 inches.

Fig. 49.—Section through adult canal and tympanum.

Fig. 50.—Section through canal and tympanum of infant at term.

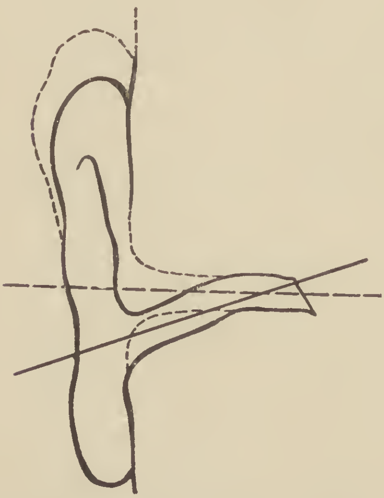

Fig. 51.—Diagram showing direction of auditory canal of adult. Dotted lines showing changed direction of canal by traction upward.

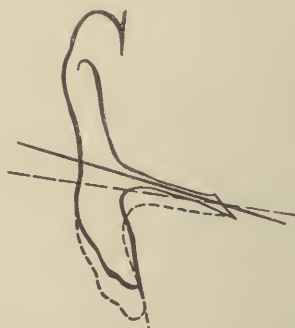

Fig. 52.—Diagram showing direction of auditory canal of infant. Dotted line showing influence of traction downward upon calibre of canal.

Use of the Mirror.—In inspecting so small a structure as the drum membrane by reflected light, one must necessarily use but one eye,—*i.e.* the eye behind the central orifice of the mirror. Both eyes, however, should be kept open, the habit, common to many beginners, of closing

the opposite eye, causing a certain amount of unnecessary eye-strain. The mirror should be adjusted with the central orifice opposite the eye nearest the source of light. That is to say, if the light is placed to the patient's left side, the mirror should be worn over the physician's right eye, and *vice versa.* As inspection of the ear frequently occupies several minutes, and tympanic operations much longer, it is important that the physician's body, head, and neck should be in a comfortable position. The light should, therefore, be thrown upon the ear and readjustments made altogether by movements and manipulations of the mirror,—the effort, common with beginners, to direct or adjust it by movements of the head usually resulting in very strained and uncomfortable positions of the neck. Having the light properly focused upon the ear, the student should devote some little time to holding it there steadily, inspecting carefully meanwhile the auricle and particularly the size and condition of the orifice of the meatus. He will make no satisfactory headway in the use of the speculum until he has the light under perfect control.

Speculum Examination.—Before practising the use of the speculum, the student may refresh his memory of the anatomical differences between the external auditory meatus in the infant and that of the adult by reference to the accompanying illustrations (49, 50, 51, 52). With adults it is often necessary to draw the pinna upward and backward in order to straighten the canal and bring the drum membrane into view. In the case of infants, on the other hand, one must usually draw the auricle somewhat downward and outward, in order to separate the floor of the canal from the roof and increase the angle between the drum membrane and the axis of the canal.

As to the size of the speculum best suited to any particular case, one should select the largest that will enter the meatus easily and without force. Nothing is accomplished beyond discomfort to the patient by the attempt to use too large a speculum. The speculum is gently introduced into the meatus and carried beyond the hairs which occasionally obstruct its lumen near its external orifice. We may then examine the different walls of the meatus for signs of acute or chronic inflammation, and for the presence or absence of obstructing substances,—*e.g.*, masses of cerumen, foreign bodies, pus, serum, etc. The latter being absent, or having been removed, we may go on to the examination of the fundus of the canal,—*i.e.*, the drum membrane (Fig. 53).

Landmarks of the Normal Drum Membrane.—For the purposes of the present study, let us assume that we are to inspect a perfectly normal and healthy drum membrane. Letting the eye follow the roof or posterior wall to the fundus of the canal, one readily recognizes the membrane by the abrupt structural and color changes which distinguish it from the surrounding walls of the meatus. As seen by reflected light, it presents the appearance of a tense membrane stretched across the lumen of the inner extremity of the canal. It varies from bluish white to ivory white in color, having a peculiar sheen, or lustre, which is one of its most constant physical characteristics in health. While the color varies somewhat

within physiological limits in different individuals, loss of lustre invariably denotes either structural changes due to chronic disease, necrosis of the surface layer of epithelial cells resulting from acute myringitis, or the deposition of some foreign substance—*e.g.*, mould (aspergilli), dried secretion, or powder blown into the canal—upon the drum membrane. The peripheral attachment to the tympanic ring (annulus tympanicus) is clearly outlined, showing the contour of the drum membrane to be irregularly ovoid (Fig. 53).

Toward the upper pole of the membrane, and nearer the anterior than the posterior margin, may be seen a small, projecting, glistening point, whiter than the surrounding structures,—usually described as of pinhead size, but in reality much smaller. Its appearance exactly portrays the actual condition,—that of a minute portion of the drum membrane pushed outward and forward into the lumen of the meatus by the small, projecting process of bone behind it,—the *short process* of the malleus (Fig. 53, *c*). This little structure, it will be remembered, lies almost in

Fig. 53.—Normal drum membrane.

the line of attachment of the anterior and posterior ligaments, or axis band, of the malleus, by which it is held firmly in position. It is the most constant landmark of the membrana tympani. It is found always in the upper and anterior part of the membrane, and is sometimes situated so far forward as to be apparently in contact with the annulus tympanicus.

Scanning now the surface of the membrane, we discover the second landmark in a more or less prominent line running directly from the short process downward and backward toward the centre of the membrane. This is the *manubrium mallei*, or handle of the malleus (*d*). The hammer handle and short process are directly continuous one with the other. Hence each is the guide to the other. Thus, if the hammer handle is first located, by tracing it upward and forward we arrive at the short process; locating first the processus brevis, we trace the hammer handle by its invariable direction downward and backward from the short process toward the centre of the membrane. Until we have located one or the other of these landmarks, we can never be positive as to what part of the drum membrane we have in view,—nor in cases of middle-ear disease can we always determine with certainty whether it is the drum membrane.

Looking now at the lower and anterior part of the drum membrane, we see a brilliantly shining triangular spot, the apex of which is located at the umbo, the opposite base being in the neighborhood of the lower and anterior arc of the tympanic ring. Its direction, therefore, from apex to base is downward and forward (*e*). This is spoken of as the *cone of light*, or *light reflex*, the latter term being not only descriptive but explanatory. It is due not to any structural change at this point, but simply to the fact that the tense membrane, drawn inward by the lower end of the hammer handle, falls normally throughout this triangular space into a plane perpendicular to the direction of the rays of light from the mirror, and, as this stream of light and the line of vision are coincident, the triangular light reflex results. Naturally, when the position of the drum membrane is changed in either direction,—*i.e.*, is either bulging or retracted,—the cone of light is either altered or lost. Alterations or absence of this landmark are, therefore, indicative of displacement, but not necessarily of any structural change in the membrana tympani.

Looking once more to the upper part of the drum membrane, we see passing almost horizontally backward from the short process a line or fold which is known as the *posterior fold of the drum membrane* (*b*). It is almost identical in position with the posterior ligament of the malleus which lies behind it. A shorter fold—so short in many cases as to be undemonstrable—runs forward from the short process to the anterior peripheral attachment of the drum membrane (*b'*). It is called the *anterior fold* of the membrana tympani. Above these folds we may sometimes make out two very fine lines, running from the short process forward and upward, and upward and backward respectively (*a*, *a'*). These lines are known as the *attachment striæ of Prussak* They pass from the short process of the malleus to the extremities of the original tympanic process or ring. Above these lines, and between them and that ungrooved part of the bony frame of the drum membrane which is formed by the outer plate of the squama, is a small space known as *Prussak's space.*[1] It is identical in position with the Rivinian notch and is inclosed by Shrapnell's membrane.

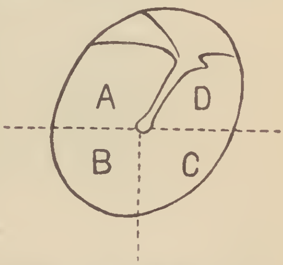

Fig. 54.—Diagram showing quadrants of membrana tensa.

Quadrants of the Drum Membrane (Fig. 54). — For convenience of description, the drum membrane is divided into four quadrants. If we imagine a vertical line passing directly downward from the umbo to the inferior margin, the drum membrane will be divided by this line and the hammer handle into two parts, an anterior and a posterior segment. Now, suppose another line, horizontal in direction, to pass through the umbo,

[1] Prussak's spaee is so called because it marks upon the drum membrane the position of a small space within the middle ear called by the same name. (See deseription on page 25.)

and the drum membrane is divided into four quadrants,—viz., the posterosuperior (A), the posteroinferior (B), the anteroinferior (C), and the anterosuperior (D).

The student or physician intending to practise otology, either as a specialty or in connection with other branches of medicine, should make himself thoroughly familiar, by examination of many ears, with the physical picture of the drum membrane as above drawn. At the same time he should bear in mind that what he sees is only part of the outer wall of the middle-ear cavity. Unless, therefore, he can supplement this picture by another, seen through the mind's eye, of the structures behind the drum membrane, his examination is robbed of half its usefulness. He should know the position and relations of the different parts of the ossicular chain so thoroughly as to be unable to look at the drum membrane without subconsciously calling to mind the relations of the deeper structures within. In recalling these relations the student will perhaps be aided by reference to Fig. 55. In the vault are located the head of the malleus and body of the incus, the former anteriorly and the latter posteriorly. In the atrium are found the short process and handle of the malleus, the long arm of the incus, and the whole of the stapes. Locating more exactly the parts of the ossicles in the atrium, we find in the anterosuperior quadrant the short process of the malleus; in the posterosuperior quadrant, the incudostapedial joint. Separating the anterosuperior from the posterosuperior quadrant is the handle of the malleus. Behind the posteroinferior quadrant of the drum-head is the niche of the round window. The importance of fixing these points clearly and permanently in mind must be apparent. Of the four quadrants of the drum membrane the posterosuperior is the one which most insistently claims our attention in tympanic disease. In acute or chronic tubal catarrh retraction of this part of the drum membrane, by pressing upon the long arm of the incus, interferes in some degree with the movements of the incudostapedial joint, with consequent impairment of hearing. In hyperplastic or sclerotic processes involving the tympanum, the newly formed connective tissue in this region may reduce the mobility of the stapes and bring about very serious loss of function. In acute suppurative otitis media, also, the posterior segment of the drum membrane is commonly most noticeably involved. It is in this region that incisions of the membrana tympani are

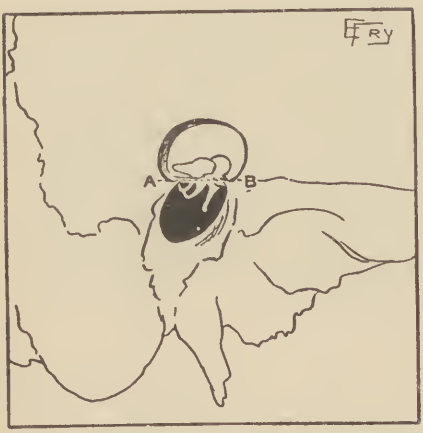

Fig. 55.—Semidiagrammatic picture, showing relations of ossicles in vault and atrium. Line A–B represents dividing line between atrium and vault.

usually indicated. Obviously this operation, often so lightly undertaken and so imperfectly executed, is not without danger in unskilful hands.

Having familiarized ourselves with its physical characteristics in health, we should now scrutinize each drum membrane examined for changes in color, position, or structure.

Color.—To the practised eye even slight changes in color are not difficult to recognize. Very considerable experience may, however, be required to enable one to determine whether moderate redness is the result of temporary congestion or is due to a subacute inflammatory process within the tympanum. The interpretation of the commoner color changes will be spoken of in a later chapter dealing with the acute middle-ear diseases.

Position.—While extreme displacement of the drum membrane in either direction is easily noted, slight changes from the normal position are quite difficult for the beginner to determine. Displacement outward, or bulging, is usually accompanied by signs of acute inflammation, and is comparatively easy to determine. The line of attachment of the hammer handle may be rendered indistinct or be completely lost to view as a result

Fig. 56.—Bulging drum membrane.

Fig. 57.—Retracted drum membrane.

of inflammatory thickening or infiltration of the drum membrane. The bulging portion of the drum membrane balloons outward into the lumen of the canal and appears nearer the examiner's eye, as it really is, than the peripheral portions (Fig. 56).

Displacement of the drum membrane inward, or retraction, is commonly unaccompanied by any of the signs of acute inflammation. The membrana tensa, carrying with it the hammer handle, is moved inward toward the promontory. The drum membrane, in being drawn or forced inward, folds itself about, or tends to form angles with, the structures to which it is attached. Hence the short process, the foreshortened hammer handle, the anterior and posterior folds, and in some cases the annulus tympanicus may appear unduly prominent (Fig. 57). While extreme retraction is easily made out even by the beginner, it requires considerable practice and experience to determine slight departures from the normal in this direction. The characteristic signs of moderate retraction will be described under tubal catarrh.

Structural Changes.—Changes in structure may take the form of uni-

form or localized thickening, of moderate or extreme atrophy of the drum membrane, of cicatrices due to old perforations, or of actual loss of continuity,—*i.e.*, perforations,—which may vary in size from a small pinhead orifice to practical destruction of the membrana tensa. Localized thickening of the drum membrane is shown usually by areas of opacity. Such opaque spots are not infrequently seen in the drum membranes of individuals whose hearing is not noticeably impaired, and are supposed in some cases to be due to calcareous deposits representing one of the tissue changes resulting from the rheumatic, or uric acid, diathesis.

Atrophy, either general or localized, is shown in some cases by abnormal thinness and transparency of the membrana tensa. In others the atrophic area appears opaque and thicker than the rest of the membrane. The latter condition represents an increase in connective tissue at the expense of the fibrous layer of the drum membrane. In such a case the atrophy is revealed only by the abnormal relaxation, or loss of tension, in the areas involved, as shown by Siegel's pneumatic otoscope to be described presently.

Perforations of the drum membrane are best observed by mapping out as nearly as possible the limits of the membrane by its peripheral attachment to the annulus tympanicus, and then looking closely for any loss of continuity in its surface within this space. As the line of demarcation between the roof of the meatus and Shrapnell's membrane is sometimes obliterated by inflammatory thickening or infiltration at this point, it is well to begin by letting one's glance travel inward over the roof of the canal and follow the drum membrane downward to its inferior margin. When the margins of a perforation are clearly defined and there is an appreciable space between the membrane and the inner tympanic wall, the form and extent of the perforation are easily determined. On the other hand, with a swollen tympanic mucosa in contact with a small and ill-defined perforation, its detection may present great difficulties to the beginner.

Fig. 58.—Rough pen-and-ink diagram of the drum membrane.

A perforation having been made out, its exact position should next be observed, and this can be determined positively only by noting its relation to either the short process or handle of the malleus. For example, a perforation behind the hammer handle must be in the posterosuperior quadrant. The student will do well to acquire early the habit of drawing diagrams of the drum membrane and noting thereon the morbid changes observed. The accompanying diagram (Fig. 58), made with a few strokes of the pen, indicates more clearly the exact size, form, and location of the perforation than could any amount of descriptive written matter.

Variations in Tension.—Conditions which cannot be determined by inspection alone are general loss of tension, relaxation of certain portions of the membrane, adhesions between the membrane and the inner tympanic wall, fixation of the hammer handle to the promontory, etc. To investigate these conditions one must depend upon some apparatus which, while leaving the drum membrane in clear view, produces alternate con-

densation and rarefaction of the air in the external auditory meatus. The Siegel otoscope (Fig. 59) admirably fulfils this need.

It consists essentially of a speculum (*a*), the expanded end of which is closed by a thin plate of glass through which the movements of the drum-head may be observed. Communicating with its interior by an aperture in the side of the speculum is a short rubber tube (*b*) the other end of which is connected with a Delstanche pump or, better, with a rubber bulb (*c*). With the small end of the speculum moistened and introduced into the auditory canal, the interior of the speculum and of the auditory meatus form practically an air-tight compartment. When the bulb is compressed, the air in the external auditory canal is condensed and the drum-head is forced inward; when pressure upon the bulb is

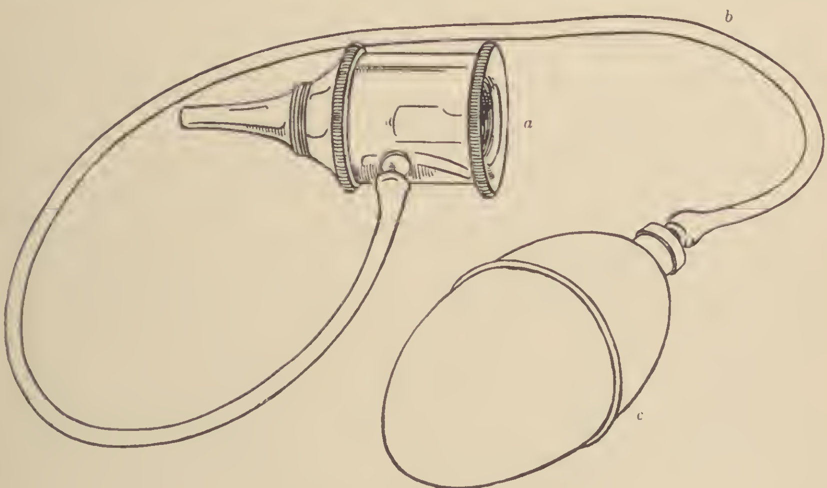

Fig. 59.—Siegel's otoscope.

released, the air in the meatus is rarefied and the drum membrane is sucked outward into the canal. Using the Delstanche pump, we produce the same mechanical results by moving the piston alternately toward and from the drum membrane. Under these manipulations the normal drum-head moves fairly evenly, the posterior segment on account of its larger surface moving rather more than the anterior. In these movements the hammer handle participates, though its excursions are less extensive and more difficult to follow with the eye than those of the posterior segment of the drum membrane.

Obviously the use of this instrument is advisable and of value only in chronic disease of the tympanum. It is equally clear that the movements of the diseased drum membrane might differ greatly from those characteristic of health, and might throw very considerable light upon the lesion with which the physician has to deal. Thus, a generally relaxed

drum membrane would execute exaggerated movements both in the anterior and posterior segments, these movement having relatively small influence upon the hammer handle. Atrophy or loss of tension, localized in certain portions of the drum membrane, would be evidenced by the ballooning of these sections, independently of the rest of the membrane, outward into the meatus. Adhesions of the drum membrane to the promontory or to any structures within the tympanum would be clearly demonstrated by its fixation at those points.

Examination of the Eustachian Tubes.—In all cases of chronic aural disease the condition of the Eustachian tubes must be determined before a correct diagnosis can be made. Inflation of the tympanum, which throws light both on the patency of the tubes and the condition of the middle ear, is therefore an essential part of the aural examination in a large class of cases. There are three recognized methods of inflation,—viz. (1) *Valsalva's method*, (2) *Politzer's method*, and (3) *inflation by means of the Eustachian catheter.*

Valsalva's method of inflation has a very limited field of usefulness. It is performed when the patient, with lips tightly closed and nostrils occluded by compression of the nose, attempts forcible expiration. By this procedure the air in the nasopharynx is condensed and finds its way under pressure through the Eustachian canals to the middle-ear cavities. This normally requires little effort, but naturally calls for greater force if the calibre of the tubes is reduced, or if the pharyngeal orifice of either tube is occluded by localized swelling or congestion in that region. It is a measure of which little use is made in otology. Patients, however, often intuitively acquire the habit of inflating the ears in this way as a means of relieving the discomfort arising from tubal catarrh, and when this is made known to us we may safely conclude that the function of the Eustachian tubes is disturbed. We may also in such cases draw conclusions as to which tube is the more occluded by requiring the patient to repeat the act and to note which ear is first inflated,—the ear last inflated corresponding, of course, to the more obstructed tube. If this ear is also the one in which the subjective symptoms are most marked, we have already some data upon which to base an opinion. Personally the author makes use of the Valsalva method of inflation in the diagnosis of one condition only, —viz., cases in which one may suspect the existence of a small perforation in the drum membrane which can not be determined definitely by inspection alone. In such a case if the patient be required to force air into the tympanum while the drum membrane is kept under view, a drop of fluid—serum or pus—will appear at the point of the perforation, or a more or less shrill whistle will result from the passage of air through the small opening in the membrane. If the drum membrane balloons outward into the lumen of the meatus without the appearance of either of these phenomena, it may be confidently assumed that no perforation exists. This, in the writer's opinion, is the one condition in which the Valsalva method is of real diagnostic value. That is to say, there is no other condition in

which all that may be learned from Valsalva inflation may not be better obtained in some other way. It is a measure which may easily become a habit with the patient to the detriment of the ear, and its general or frequent use is to be advised against.

Politzer's method of inflation resembles that of Valsalva in that both depend upon condensation of the air in the nasopharynx. Since this procedure was first proposed, the universal recognition of its value and its general adoption by otologists of all countries have resulted in some slight modifications in the instruments employed.

Politzer's inflating apparatus (Fig. 60) consists of a compressible, soft rubber air-bag communicating at one end with a piece of rubber tubing the end of which fits over a hard rubber tubular nozzle, or nose-piece.

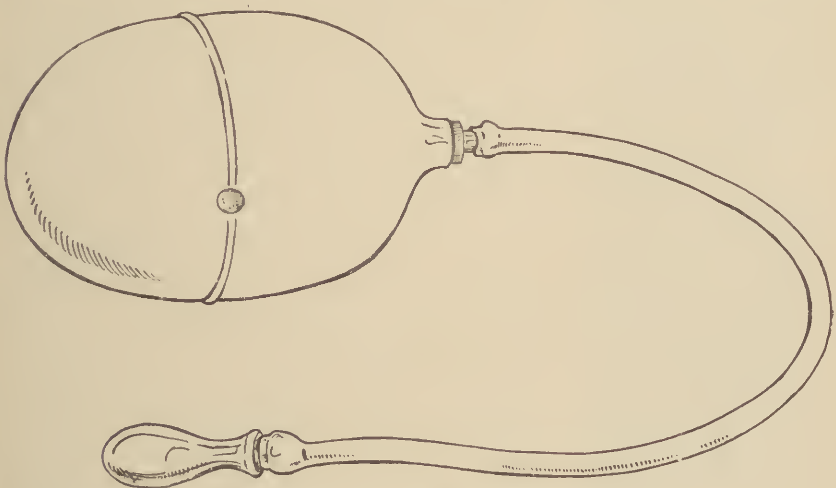

Fig. 60.—Politzer's inflating apparatus.

The air-bag is provided with a valve by means of which, after the contained air has been expelled, a fresh supply is taken in without the necessity of removing the nozzle from the patient's nose.

Technic.—The patient and physician are seated opposite each other. The former is given a glass of water and told to take a sip of it into his mouth and to swallow it at a signal from the physician. With his left hand the physician now inserts the nozzle of the inflating apparatus into the patient's nostril corresponding to the ear he wishes to examine, at the same time closing the nasal passages by pressure upon both alæ nasi. The air-bag meanwhile is held ready for use in the physician's right hand. The patient is now told to swallow and, synchronously with this act on his part, the air-bag is quickly and forcibly compressed. The act of deglutition brings the soft palate into contact with the posterior wall of the pharynx and effectually closes the nasopharynx. The air in the naso-

pharynx is at the same time condensed by the influx of additional air from the inflating bag, and forces its way through the Eustachian tubes into the tympanic cavities. By reason of the valvular opening in the air-bag, inflation by this method may be repeated several times without removing the end-piece from the patient's nose. A modification of Politzer's method is by requiring the patient, instead of swallowing water, to repeat some word ending in K,—thus, hock, hock, hock, etc.,—the lingering enunciation of this guttural as a terminal sound serving to hold the soft palate in contact with the posterior pharyngeal wall and thus shut off the nasopharynx from the laryngopharynx below. The first method, however, has the advantage that the act of swallowing not only closes the nasopharynx but also serves to separate the anterior from the posterior wall of the membrano-cartilaginous tube, and thus facilitates the freer passage of air to the tympanum. Another method is by requiring the patient, after the tip of the inflating apparatus has been properly adjusted, to close the lips tightly and forcibly balloon out the cheeks. This, of course, does not close the nasopharynx, but serves to condense the air in the entire respiratory tract. Obviously the pressure of additional air from the air-bag would tend to force the patency of the tubes. It seems to be a combination of the Valsalva and Politzer methods. It is very useful in dealing with certain children who will not co-operate with the physician in carrying out the method as proposed by Politzer.

FIG. 61.—Eustachian catheter (silver).

Politzer's method of inflation has a wide field of usefulness. As a diagnostic measure its position is distinctly secondary to inflation by catheter. It must be used, however, in certain classes of cases, among which are the following:

(1) Young children, who will rarely submit to catheterization.

(2) Very nervous adults who in some cases (rare) will not submit to the use of the catheter at the first sitting.

(3) Adult patients in whom nasal abnormalities or occlusions contraindicate or render impossible the use of the catheter (rare).

Inflation per Catheter.—In the writer's opinion the catheter offers by far the most satisfactory and reliable method of examination in the great majority of cases. Its full usefulness, however, requires a degree of technical skill and experience in interpreting the sounds heard through the otoscope, which can be acquired only after long practice. The student of practical otology can not, therefore, too carefully or persistently practise its use. If at the end of a year's practice in a busy clinic he has acquired considerable skill in catheterization, he will assuredly be yet more skilful at the end of a second year. By skill is meant not merely the ability to find the tubal orifice, but to accomplish this with the least mechanical irritation of the nasal and pharyngeal mucosa, and therefore with minimum injury and discomfort to the patient. As it is absolutely essential that the necessary instruments be of right material and design, a few words may well be devoted to them here.

The Catheter.—The accompanying figure (Fig. 61) clearly illustrates the correct size and form. The expanded outer end (*a*) receives the tip or end-piece of the inflating apparatus. The small end (*b*) must be smooth,—*i.e.*, without sharp edges which might injure the nasal or tubal mucosa. The curve near the end enables the physician, after it has passed into the cavity of the nasopharynx, by rotating the catheter, to bring the beak into the Eustachian orifice. While the curve indicated in the illustration is the one best suited to a majority of cases, it obviously must be changed somewhat to meet the anatomical requirements of certain pharynges. The catheter should therefore be made of malleable silver to allow of such changes as may be required. The ring (*c*) is of use as a guide by which in a case presenting anatomical difficulties the surgeon may know in what direction the beak is pointing. As to length, the writer uses and prefers a catheter of 5¾ inches. It should not exceed 6 inches. Any unnecessary length—*i.e.*, which leaves more of the catheter outside of the nose than is necessary for its manipulation—simply interferes with its proper control and adds to the patient's discomfort. A catheter longer than 6 inches has never in the writer's experience been found necessary. The catheters come in three sizes, or calibres, of which the illustration represents the intermediate, and the one indicated in the great majority of cases. The largest is practically never used by the writer. The smallest is necessary in some cases.[2]

The Dench inflating apparatus (Fig. 62) is undoubtedly the best so far devised for catheter inflation. Between the air-bag (*a*) and the tip (*c*) which fits into the outer end of the catheter, is a hard-rubber bottle (*b*). This bottle enables one to employ medicated vapors in the treatment of the tubal and tympanic mucosa. When the stopper is in the position

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[2] Catheters of hard rubber, described in some text-books, are to be condemned, if for no other reason, because they can not be thoroughly sterilized. Catheters of hard "German silver" offer the decided disadvantage that they can not be bent. Catheters with bulbous ends do not admit of as fine adjustment as those of smaller terminal calibre, but may offer distinct advantages as practice instruments for beginners.

shown in the illustration (*d-e*), air expelled from the air-bag does not pass through the bottle and is therefore not influenced by its contents; but when the stopper is turned so as to occupy the position indicated by the line *f-g*, the air must pass through the bottle before reaching the catheter and is in this way impregnated with any volatile drugs—*e.g.*, alcohol,

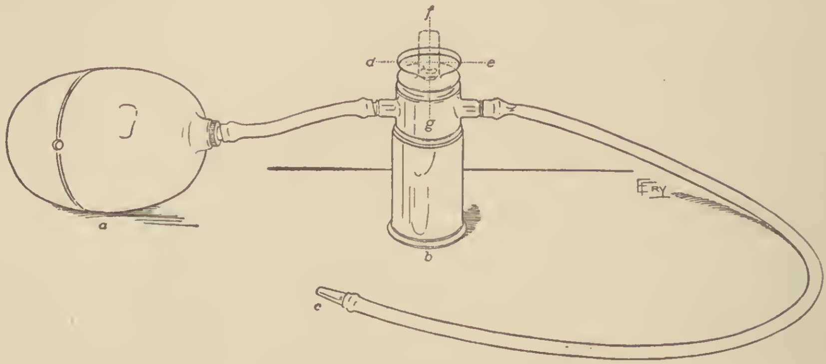

Fig. 62.—Dench inflating apparatus.

solutions of menthol, camphor, iodine, etc.—therein contained. The rubber tubing prevents the movements of the air-bag from being communicated to the catheter. The valvular opening into the air-bag enables the physician to repeat the act of inflation any number of times without moving the catheter.

The Otoscope or Diagnostic Tube (Fig. 63).—This is simply a piece of rubber tubing, about 30 inches long, into each end of which is fitted a small hollow ear-piece somewhat similar to, but smaller than, the ear-tips

Fig. 63.—Diagnostic tube.

of the ordinary stethoscope. Usually one is of white bone and the other of hard black rubber. This enables one to distinguish them,—an obvious advantage in a busy clinic, where the aurist may be pardoned for wishing to reserve one end for himself, leaving to his patients the exclusive use of the other. Inserting one end into his own ear and the other into that of his patient, he can during inflation obtain fairly accurate information as

to the condition of the tube and tympanum by the auscultatory signs obtained through the otoscope. With a normal tube and tympanum, he will hear during inflation a rather low, blowing sound giving somewhat the impression of a sound originating in his own ear. While usually low, the pitch will of course vary somewhat with variations in the calibre of different tubes.

Of abnormal conditions which may be made known to us by the otoscope, may be mentioned the following:

(a) *Absolute occlusion of the tube* as shown by absence of all sound of air entering the tympanum. Obviously this sign is without value unless the physician has sufficient technical skill to feel sure that his catheter is properly placed.

(b) *Patent but abnormally narrow tube,* shown by clear sound of unusually high pitch.

(c) *Abnormally wide tube,* made known by very low-pitched, rough sound and the absence of any resistance to the compression of the air-bulb.

(d) *Presence of excessive mucus or other fluid secretion within the tube,* shown by small, intermittent râles, finally ceasing as the moisture is distributed or blown into the nasopharynx by the return air current.

(e) *Presence of fluid in atrium,* shown by more or less constant, bubbling râles, giving the impression of air passing through fluid,—the actual physical condition.

(f) *Abnormal relaxation of the drum membrane,* as shown by certain characteristic sounds due to the flapping outward of the drum membrane during compression of the air-bag, and its recoil as the air pressure subsides.

(g) *Small perforations of the drum membrane,* made known by whistling, squeaking, or singing sounds due to vibration of the edges of the perforation.

(h) *Large perforations in, or absence of, the drum membrane,* made known by the sensation of air blowing against the physician's drum membrane.

It is quite useless to attempt to describe more minutely than as given above the various sounds and signs obtained through the otoscope during inflation. With practice and careful study of cases, these sounds will soon rank among the most valuable guides as to the conditions present.

Technic.—The physician is seated directly opposite his patient, with the ends of the diagnostic tube adjusted in his own and the ear to be inflated (Fig. 64). The patient is directed to hold the head with chin slightly depressed, to keep the mouth closed, to continue breathing quietly through the nose, and to resist the impulse to jerk or move the head backward and out of the physician's reach. He is asked to hold the bottle of the inflating apparatus, this arrangement leaving the air-bag in convenient reach of the physician's hand. To be in proper relation to the mouth of the tube, the catheter must reach the nasopharynx by way of the inferior meatus of the nostril corresponding to the ear to be inflated (Figs. 65 and

FIG. 64.—Position of physician and patient during catheter inflation

66). At the start the catheter is held between the thumb and forefinger of the right hand, the tip of the inflating apparatus being held between the third and little fingers of the same hand, ready for introduction into the outer expanded end of the catheter as soon as the latter shall have been placed in proper position. Holding the catheter at first almost in the vertical position, with the curved end pointing forward, the tip is introduced into the inferior meatus of the nostril corresponding to the ear to be inflated. In order to insure against its entering the middle meatus, it is now quickly elevated from the vertical into the horizontal position as it enters the nostril, the tip being kept in light contact with the nasal floor, and is carried directly backward until it is felt in very light contact with the posterior pharyngeal wall. It is next rotated outward toward the ear until the guide ring points in a direction about midway between the horizontal and vertical planes. It is then withdrawn slightly until its beak is arrested by the anterior wall of the fossa of Rosenmüller, which, it will be remembered, corresponds in position with the posterior wall of the Eustachian orifice. Over this elevation it is now lightly drawn, the catheter being then rotated further outward and upward until the guide ring points somewhat upward, describing an angle of 30 or 35 degrees with the horizontal plane. This should bring its extremity into its proper position in the pharyngeal mouth of the tube. The outer end of the catheter is now transferred to the left hand and the tip of the inflating tube carefully inserted.[3] The right hand is thus free to grasp the air-bag, and the difficult part of the procedure is accomplished. The sounds heard through the otoscope now furnish additional information as to the exact position of the catheter within the Eustachian orifice, and enable one to make such slight readjustments as are necessary to give the best inflation.

Other methods of placing the catheter have been described,—as, for instance, (1) by passing the catheter as before, through the inferior meatus until its beak touches the posterior wall of the pharynx, rotating it toward the opposite ear, withdrawing it until its curved end is engaged by the posterior margin of the nasal septum, then rotating it downward and outward in the opposite direction until its beak enters the Eustachian orifice; (2) passing the catheter backward with beak resting on the nasal floor, noting when the beak sinks into the nasopharynx and then rotating it directly into the orifice of the tube to be inflated. These are distinctly less reliable than the method first described, which is the one which should be practised by the student of practical otology. By so doing he will surely in time acquire a facility which will enable him to dispense with

[3] Some aurists prefer and advise that the tip of the inflating apparatus should be securely inserted into the outer end of the catheter before the latter is introduced into the patient's nose. This prevents the discomfort which might arise during its adjustment after the catheter is in position. In the writer's experience a more delicate manipulation of the catheter has seemed possible by the method above advised. A little practice will enable one to make the connection between the catheter and the inflating apparatus without discomfort to the patient.

some of the steps described. Thus, the practised aurist learns to ignore the fossa of Rosenmüller as a guide, and intuitively withdraws the catheter to the right position opposite the mouth of the tube. The student, however, will do well to follow literally the steps here described, which alone will lead to the degree of technical skill which robs this procedure of its discomfort to the patient.

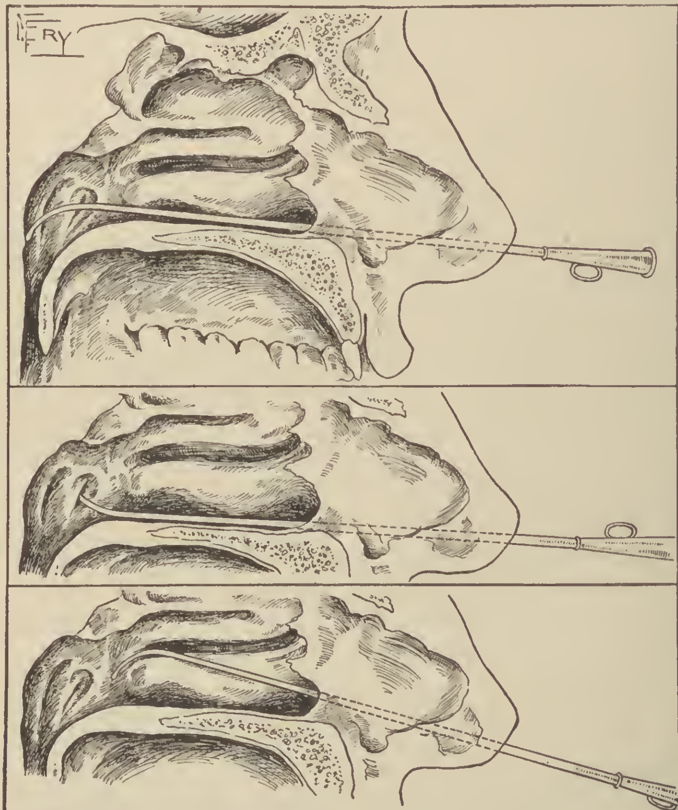

Fig. 65.—Correct pathway of catheter to posterior pharyngeal wall.
Fig. 66.—Correct position of catheter within tubal orifice.
Fig. 67.—Incorrect introduction of catheter,—*i.e.*, through middle meatus.

There are one or two points mention of which may be of use to the beginner. He should learn at the start to recognize surely the sound which comes to him through the otoscope when the end of the catheter is not in the mouth of the tube but in the fossa of Rosenmüller. This sound is loud, rough, does not have the character produced by air passing through an open tube, and does not sound as if originating in or near the physician's own ear. Unless this sound is definitely recognized, the student may waste considerable time in the mistaken belief that he is prac-

PLATE IV.

FIG. 68.—Tongue depressor (A). FIG. 69.—Nasal speculum (B). FIG. 70.—Nasal speculum (C). FIG. 71.—Laryngeal mirror (D). FIG. 72.—Cotton applicator (E). FIG. 73.—Nasal probe (F).

tising inflation while in reality his catheter may never have entered the Eustachian orifice. When properly placed, the catheter is held in position by slight pressure of the outer end against the nasal septum, this tending to throw the other end, or beak, in the opposite direction and further into the Eustachian orifice. If now the patient complains or shows signs of pain, learn from him whether the pain is referred to the nose or the throat. If in the nose, slight shifting of the position of the catheter here or easing of the pressure will usually relieve the discomfort. If, however, the pain is referred to the throat, it is due often to rotation of the catheter too far in one or the other direction. A little care in correcting these minor errors of technic will render this a bearable, if never a pleasant, experience to the patient.

Surgical Emphysema.—There is one accident which must be mentioned as a possible result of careless catheter inflation,—viz., traumatic emphysema. It occurs when air from the inflating apparatus is propelled through some false passage into the tissues surrounding the membrano-cartilaginous tube. It probably always indicates either an abrasion of the tubal mucosa, or an incorrect position of the catheter, as a result of which air is forced through the mucous membrane into the tissues beneath. The accident usually gives rise to very sharp and sudden pain, which is, therefore, an indication that the inflation should be immediately discontinued. The diagnosis is confirmed when palpation of the tubal region—*i.e.*, between the tip of the mastoid and the angle of the jaw and downward along the anterior border of the sternomastoid—is accompanied by distinct crepitus. While painful and often alarming to the patient, the condition is not dangerous, the air being usually absorbed within from 24 to 48 hours. The patient should, therefore, be assured that the symptoms will disappear spontaneously within a day or two. If treatment is called for, very gentle massage may help to expel the air from the tissues.

The Nose, Nasopharynx, and Throat.—The relation of the ear to the nose and nasopharynx is so intimate, and the diseases of the two regions are so interdependent, that one must assume for the special student of otology some preliminary or collateral study of rhinology and laryngology. No examination of the ear is complete until one has carefully inspected the nose and nasopharynx. The author will speak very briefly of this region, dealing only with what he believes to be absolutely essential to a complete aural examination.

A forehead mirror with focal distance of from 12 to 15 inches gives very satisfactory illumination. In addition to the mirror, the instruments necessary in an examination of the nose and pharynx are a tongue depressor (Fig. 68), a nasal speculum (Figs. 69, 70), a postnasal mirror (Fig. 71), cotton applicators (Fig. 72), and a nasal probe (Fig. 73).

One saves time by observing some regular order in one's examination, and the author prefers to examine first the nasal cavities, then the posterior wall of the pharynx and the faucial tonsils, and last the nasopharynx, or postnasal space.

Examination of the Nose.—From the view-point of the aurist the examination of the nose is chiefly for the purpose of determining any condition within the nasal cavities which may predispose to, or directly excite, nasopharyngeal congestion and catarrh of the Eustachian tubes.

The conditions to be looked for are: (1) *Abnormalities of the nasal septum,—e.g.* (a) deflections to one or the other side. This may be very slight or so marked as completely to occlude one nostril. It may be limited to the anterior cartilaginous portion, or include the posterior or osseous part of the septum, involving the vertical plates of both the vomer and ethmoid. (b) Localized septal ridges or spurs, which again may be confined to the cartilaginous part (ecchondroses) or to the posterior bony portion (exostoses), or the ridge may include both, running from before backward and upward along the line of junction of the vomer to the cartilage in front and to the ethmoid plate behind. (2) *Hypertrophy of the turbinated bodies.* By anterior rhinoscopy one can usually see only the anterior half of these structures. If one or both inferior turbinates are noticeably enlarged, we should determine by palpation whether the increase in size is due to an actual hyperplasia—*i.e.*, depositions of new connective tissue—or to engorgement of the turbinal vessels. In the former case the turbinal body offers considerable resistance to compression by means of the nasal probe, and its size is only moderately reduced by the application of adrenalin or cocaine. When the enlargement is due to vascular engorgement, the turbinate pits deeply on pressure but quickly regains its former size as soon as the compressing probe is removed, and it shrinks enormously under the influence of the suprarenal preparations.

The middle turbinates are not quite so easily inspected. When the septum is deflected to one or the other side, it may be quite impossible to see the middle turbinate on the side to which the septum is bent, without first shrinking the corresponding inferior turbinal body. Usually, however, by means of a proper speculum and by tilting the patient's head somewhat backward, both middle turbinates may be brought into view. Whenever either of these bodies are noticeably enlarged, the corresponding nasal cavity should be thoroughly exsanguinated by means of adrenalin chloride. This aids in determining to what extent, if any, the enlargement is due to simple congestion. The nasal cavity should now be carefully inspected for nasal polypi and for a possible escape of pus from the middle meatus,—*i.e.*, from beneath the middle turbinate. Either of these conditions might point to disease involving either the frontal sinus or the anterior ethmoid cells, or both.

The Throat.—In examining the throat, one should endeavor to train one's eye to observe quickly, prolonged use of the tongue-depressor causing discomfort, reflex pharyngeal irritation, and often retching. This may render subsequent examination of the postnasal space unsatisfactory or impossible. The tongue-depressor should be introduced into the mouth only so far as is necessary to press upon the most elevated and arched portion of the dorsum of the tongue,—pressure too far back causing re-

flex irritation and "gagging," or retching. With the tongue depressed, one should note quickly first the size of the faucial tonsils,—*i.e.*, whether they are of normal size or hypertrophied. As to the standard by which the size is to be judged, so distinguished an authority as Bosworth is of the opinion that whenever the tonsil is demonstrable by inspection, it should be regarded as representing an abnormal growth. The more commonly accepted view is that any projection of the tonsil toward the median line so as to encroach, however slightly, upon the cavity of the pharynx is abnormal, and represents hypertrophy. It must be remembered that tonsils but slightly enlarged while the patient is in his usual health may be subject to great increase in size under the influence of any nasopharyngeal congestion or inflammation,—*e.g.*, a common "cold." Moderate hypertrophy of the tonsils in adults may, however, be absolutely without influence upon the ears. In young children, on the other hand, any considerable enlargement of the faucial tonsils is almost invariably associated with the presence of pharyngeal adenoids. Tonsillar hypertrophy has, therefore, a very decided bearing upon the condition of the ears in childhood.

On the posterior pharyngeal wall one should note the presence or absence of the following conditions,—viz.: (a) Dilatation of the superficial veins, a condition which in adults very frequently results from nasal obstruction and the irritation due to mouth-breathing during sleep. (b) The so-called granular pharynx, characterized by the presence of granulations upon the pharyngeal wall. They are usually most noticeable along the median line of the pharynx just behind and below the uvula. This condition in children almost invariably indicates the presence of nasopharyngeal adenoids. (c) The presence of dried or tenacious secretion adhering to the pharyngeal wall. With this is frequently seen a more or less glazed condition of the pharyngeal mucosa, a condition characteristic of atrophic rhinopharyngitis. (d) The physical signs of acute pharyngitis.

Posterior Rhinoscopy.—The examination of the postnasal space by means of the small rhinoscopic mirror forms an important part of the examination of adult patients suffering from aural disease. It consists of depressing the tongue by means of a tongue-depressor, and inserting a small mirror backward over the tongue, under and behind the soft palate, so that by reflected light one may obtain a clear view of the roof of the pharynx, the posterior margin of the nasal septum, the posterior ends of the middle and inferior turbinal bodies, the fossæ of Rosenmüller, and the pharyngeal orifices of the Eustachian tubes. Here, even more than in examining the posterior pharyngeal wall and faucial tonsils, it is important to acquire the habit of quick but accurate observation. Of possible abnormalities bearing upon an existing aural disorder, one should look especially for the following conditions,—viz.: (A) *Hypertrophied lymphoid tissue upon the roof of the nasopharynx.* An adenoid growth in this region, if of considerable size and located well forward, will inevitably cut off from view the upper part of the nasal septum. If the whole of the posterior

margin of the septum can be seen, and the roof of the pharynx arches evenly above this without noticeable obstructing mass, the cause of an existing nasal or aural disorder must be looked for elsewhere. (B) *Lymphoid hypertrophy in the fossæ of Rosenmüller.* In some cases—and particularly where previous operations for the removal of adenoids have been performed—the pharyngeal roof may be practically clear yet considerable masses of adenoid tissue are found behind the Eustachian orifices in the fossæ of Rosenmüller. This condition is particularly likely to induce morbid changes in the middle ear, leading to permanent impairment of hearing. (C) *Hypertrophy of the posterior ends of the inferior turbinal bodies.* This condition is nearly always dependent upon some other lesion in the anterior nares. (D) *Congestion, or swelling, of the tissues about the Eustachian tubes.* This is often quite noticeably present during acute tubal catarrh, and is usually dependent upon some coexisting nasal or nasopharyngeal lesion.

Any of the above conditions would necessarily have some bearing upon an aural disorder. Their presence or absence is best determined in adult patients by posterior rhinoscopy. With young children, on the other hand, the use of the postnasal mirror is in most cases impossible, and we are obliged to depend upon digital examination. The technic of examining the postnasal space by reflected light has been so fully described in most text-books on diseases of the throat and nose, that its repetition here hardly seems necessary.[4] Inspection of this part of the respiratory tract forms, however, a very important part of every aural examination in adults, and should be carefully practised by every aurist who would do thorough and effective work.

Obviously there are many cases—including all cases of non-suppurative aural disease—in which the examination would be altogether unsatisfactory without careful testing of the patient's hearing power. This, however, is a subject of such importance in otology that it seems better to consider it in a separate chapter.

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[4] A clear and satisfactory statement of the technic of this procedure and the difficulties it may present will be found in Coakley's "Manual of Diseases of the Nose and Throat."

CHAPTER III.

FUNCTIONAL EXAMINATION OF THE COCHLEAR APPARATUS; HEARING TESTS.

To THE average practitioner of medicine the functional examination of the ears is an unknown art. If he has kept his medical knowledge intact as he received it from the medical school, he should be in a position to make a practical differentiation between the commoner nerve lesions. But if his patient suffers from impaired hearing, he can not by his own efforts determine whether this functional loss is due to an attack of acute tubal catarrh or to a lesion involving the labyrinth or auditory nerve. Nor is it an easy matter for him to gain from otological literature or text-books a practical working knowledge of the more important hearing tests, their rationale and the proper method of applying them. The author wishes, therefore, if possible, to present the essentials of this important subject in a form more easily available both by the busy physician and by the overworked medical student.

Before we can draw any practical conclusions from a functional examination, we must have in mind some normal standard of hearing with which to compare results. It will be remembered that the normal ear appreciates musical tones between two extremes of pitch,—the tone produced by a sonorous body executing 18 double vibrations per second representing approximately the lower limit of normal hearing, and 41,000 double vibrations per second representing the upper tone limit.[1]

Air Conduction and Bone Conduction of Sound.—Sound is brought to the organ of hearing in two ways,—(1) by *air conduction,* as when the vibrating body is at a variable distance from the ear, the sound waves being transmitted through the medium of the surrounding air; and (2) by *bone conduction,* as when the vibrating body is in contact with the skull, the sound waves being propagated through the solid medium of the cranial bones. Rinné demonstrated in 1855 that the human ear in health appreciates a given musical sound—*e.g.,* that produced by the C tuning-fork (128 d. v.)—twice as long by air conduction as by bone conduction. This fact may be demonstrated in the following way: the fork having been thrown into maximum vibration, place the end of its handle in contact

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[1] There can be no doubt that slight variations both as to the upper and lower tone limits may occur in different normal individuals. These variations possibly explain in part the different conclusions of different observers. Thus, Bezold, as a result of his examination of a large number of normal ears, placed the lower limit at 12 double vibrations per second. Helmholtz gave 32,500 double vibrations per second as the upper limit of possible tone perception. Politzer places it at 40,000 double vibrations, and Bezold at 41,000 double vibrations per second. 41,000 double vibrations correspond to the sound produced by the Edelmann-Galton whistle shortened to 0.5 and this may be accepted as the upper tone limit for the human ear.

with any portion of that side of the skull corresponding to the ear to be examined. The sound will be conveyed to the ear by means of the cranial bones. Request the person upon whom this test is made to indicate by raising his hand the exact moment, as nearly as he can determine it, at which the tone is lost to him. Now remove the fork from the skull and, without setting it in fresh vibration, bring the ends of the still vibrating prongs opposite the auditory canal of the ear to be examined. The tone will be again distinctly heard, and will continue to be appreciated during a period about equal to that during which it was heard through the cranial bones. In other words, it is heard by air conduction about twice as long as by bone conduction. This approximately 1-to-2 ratio between bone conduction and air conduction is one of the most constant functional characteristics of the normal ear, and is changed only in certain forms of aural disease.

Normal Hearing Distances for the Watch-tick, the Acoumeter, the Conversational Voice and Whisper.—The experience of many observers in making a large number of hearing tests in persons with normal ears has established certain hearing distances for particular sounds as representing approximately the normal standard.

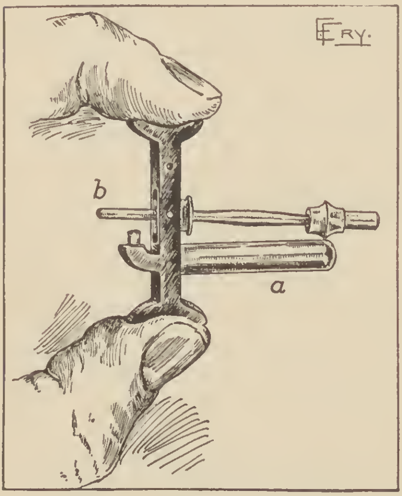

Fig. 74.—Politzer's acoumeter.

The Watch-tick.—This is a popular hearing test with the laity and medical profession alike. Naturally this sound varies considerably in intensity with the size, form, thickness of covers, etc., of different watches. Taking, however, a man's watch of average size, its tick will be heard by the normal ear of a young adult—say, of twenty to thirty-five years—at a distance of 40 to 50 inches. This hearing distance is subject to considerable variations within physiological limits, 30 inches not necessarily implying impaired hearing, and 60 inches falling within the hearing range of some individuals. As age advances, the hearing distance for the watch is gradually diminished, and in old age—*i.e.*, after sixty years—may be completely lost in individuals who, nevertheless, retain sufficient hearing power for the ordinary purposes of life. It is not always a reliable test of the usefulness of the organ, many persons whose hearing for the watch-tick is much below par having apparently no difficulty in interpreting the voice sounds in conversation.

Politzer's Acoumeter.—This very useful little instrument is so clearly shown by Fig. 74 that a detailed description is hardly necessary. The horizontal metal bar (*a*) is immovably attached to the upright column

of hard rubber. The upper rod, or hammer, is movable so that when the short end (*b*) is depressed, the hammer is raised, and when the short lever is released, the hammer falls of its own weight, and, therefore, always with the same force, upon the metal bar. It produces a clicking sound somewhat similar to the watch-tick, but very much louder. The different parts are of prescribed size and length, so that different instruments are supposed to produce approximately the same intensity of sound. The results of many tests have established 45 to 50 feet as about the normal hearing distance for the acoumeter.

The Conversational Voice and Whisper.—This is the final test of the patient's hearing power as a means of communicating with the world of practical affairs. It is, therefore, the test in which he is most vitally interested. In making this test, the physician and patient stand at opposite ends of the room, the ear to be examined being turned toward the physician. The opposite ear is closed by a finger pressed firmly into the meatus. Standing thus sideways toward the physician, the patient can not see his lips, and the element of lip-reading is eliminated. The physician now repeats the words or numbers which he wishes to employ, the patient having been instructed to repeat them after him. If the patient can not hear, or hesitates or calls the words incorrectly, the physician at once moves nearer and repeats the experiment, using different words, but those having as nearly as possible the same sound values. The distance between patient and physician is thus reduced until one is reached at which the words are repeated promptly and correctly. This is estimated in feet and carefully noted upon the test card.

Owing to the difficulty in commanding always the same tone and intensity of voice, we are accustomed to test also with whispered words. In whispered speech, tone or pitch variations are practically eliminated, and it is much easier to cultivate a uniform intensity. Thus, one may employ a very low whisper, which can barely be heard by the normal ear at a few feet from the ear, or a very loud whisper, as when one partially fills one's lungs and speaks the words without voice as forcibly as possible. Between the low and the forced whisper, a moderate whisper may be employed, which with a little practice may be made nearly, if not quite, of uniform intensity. Words spoken in the forced whisper can be heard by the normal ear at a greater distance than the same words spoken in a moderately low conversational voice, and the moderate whisper of one who has practised its use can probably be heard by the perfect organ of hearing at about the same distance as the moderately low speaking voice. Considerable time and effort have been expended by many distinguished students of otology in an attempt to determine the normal hearing distance both for the conversational voice and for whispered speech. Owing to variations in pitch, timbre, volume, etc., in the voices of different individuals, and also to possible differences in clearness of enunciation, such questions can not be determined exactly. Making allowance for such voice variations, however, it may be said, probably with approximate

accuracy, that the average conversational voice will be comprehended by the normal ear in a perfectly quiet room or hall at a distance of from 60 to 70 feet, and that the whisper of moderate force will be heard at about the same distance.

Recapitulation.—Defining briefly the functionally normal ear, we may say that an ear which hears the watch tick at a distance of 35 to 50 inches, and the acoumeter 40 to 50 feet; which hears and correctly interprets the conversational voice and the whisper of moderate force at a distance of 60 to 70 feet; which appreciates musical tones ranging between 18 double vibrations and 41,000 double vibrations per second; and which hears a vibrating tuning-fork of 128 double vibrations, C, twice as long by air conduction as by bone conduction, is normal so far as its function is concerned.

Before taking up in regular order the various tests by which we endeavor to determine the site of an aural lesion, I wish to speak briefly of certain functional changes which occur quite regularly in advanced tympanic disease, and of others which are equally characteristic of the deafness caused by labyrinthine or auditory nerve lesions. These functional changes are few in number; but once they are accepted as typifying the two main divisions of aural disease (*i.e.*, tympanic and labyrinthine), it will be seen that the various functional tests are for the most part but convenient methods of establishing the presence or absence of changes the significance of which we already know.

Changes in the Tone Limits.—Loss or Impairment of Hearing for the Lower Musical Tones.—It is a fact well known to otologists that lesions confined to the sound-conducting apparatus are almost invariably accompanied by some loss of hearing for the lower tones of the musical scale while the perception of high musical tones may be in no way interfered with. A very simple experiment will enable us to demonstrate this phenomenon as characteristic of tympanic disease. Ask a person with normal hearing to close both ears with a finger placed in each meatus. Now bring a vibrating tuning-fork of low pitch—*e.g.*, of 26, 32, 36, 40, 43, or 48 double vibrations—in front of the auricle. Its sound will not be heard. Repeat this experiment with the C^{-1} fork (64 d. v.) and its tone can not be excluded, while forks of still higher pitch—*e.g.*, c^1 (256 d. v.)—are heard quite loudly in spite of any effort to close the external auditory canals. This loss of hearing for the lower musical tones occurs in stenosis of the Eustachian tubes, in chronic catarrhal otitis media, or as a result of occlusion of the external auditory meatus. In other words, lesions involving any part of the sound-conducting mechanism interfere with the perception of low musical tones, the hearing for the high notes being affected comparatively little thereby. This phenomenon may seem a little more comprehensible if we bear in mind that very low musical tones represent long sound waves with slow vibrations of relatively great amplitude, whereas tones of high pitch are produced by short sound waves with rapid vibrations of very small amplitude. It is conceivable that mechanical

obstacles to the movements of the ossicular chain would interfere first with the slower and greater excursions necessary to the transmission of the lower tones, and only in very advanced stages of ossicular fixation with the very rapid but slight excursions required to transmit the high musical tones. In *elevation of the lower tone limit,* or impaired audition for the lower musical tones, then, we have the first functional change which is characteristic of disease of the sound-conducting mechanism.

Loss or Impairment of Hearing for the Higher Tones of the Musical Scale.—While not necessarily or invariably present, diminution or loss of hearing for the extreme upper tones of the musical scale occurs with sufficient regularity in diseases of the labyrinth or auditory nerve to justify a diagnosis of labyrinthine involvement in a majority of cases. This is rather difficult to understand, since it would at first seem that morbid processes within the labyrinth would be likely to involve any of the cochlear structures. It is known, however, that that portion of the basilar membrane which has to do with the perception of the higher musical sounds is situated in the lower part of the cochlea,—*i.e.,* in that part which is in most immediate relation to the foot-plate of the stapes and the membrane of the round window. It is believed that in most labyrinthine disorders (*i.e.,* other than congenital deafness or deaf-mutism) this part of the cochlea and membrana basilaris is most frequently involved, and that the perception of the high tones is thereby interfered with. However this may be, it is a fact which clinical experience has abundantly confirmed, that with acquired lesions of the inner ear some loss of hearing for the upper end of the musical scale is almost invariably present. The author has repeatedly made functional tests in patients to whom half the notes of the Galton whistle were totally inaudible, whose lower limit was only moderately changed. Here, then, in *lowering of the upper tone limit* we have a counter functional change which points quite strongly to disease of the labyrinth, and we are prepared for our first step toward a differential diagnosis.

Changes in the Period of Hearing by Bone Conduction.—Increase.—To Schwabach is due the credit of having definitely established the fact—already partially demonstrated by Rinné—that in disease of the sound-conducting apparatus the period of hearing by bone conduction is distinctly prolonged. This may be easily demonstrated by the following simple experiment: holding a vibrating tuning-fork in contact with the skull of a person of normal hearing, ask him to indicate by a motion of his hand the moment at which the tone is no longer perceived. Now, without removing the fork from the skull, require him to close both ears by a finger placed in each external auditory meatus. The sound of the still vibrating fork will again be distinctly heard. In other words, we have created the mechanical equivalent of an obstructive lesion of the conducting apparatus with the result that bone conduction is increased.

The explanation of the increase in hearing by bone conduction in tympanic disease proper is not altogether clear. Bezold believed that in

bone conduction the sound is not transmitted directly through the cranial bones to the structures of the cochlea, but is conducted through bone to the drum membrane, or at least to the foot-plate of the stapes, and is transmitted thence to the labyrinth; in other words, that it is distinctly an *osteo-tympanic* conduction. In explanation of the increased bone conduction regularly resulting from tympanic disease, he advanced the following hypotheses: (1) That normally the whole conducting chain—drum membrane, ossicles, and annular ligament connecting the foot-plate of the stapes to the rim of the oval window—is in a state of perfectly mobile equilibrium. (2) That any abnormal or pathological condition within the tympanum, either by changing the position of the stapes or inducing inflammatory changes in the region of the oval window, places the fibres of the ligamentum annulare in some degree on the stretch. (3) That stretching of the annular ligament interferes with the transmission of sound waves reaching the ear by air conduction, but distinctly favors the propagation of sound waves through the cranial bones to the foot-plate of the stapes. According to this theory it is also obvious that ankylosis of the stapes due to hyperplasia of connective tissue about the oval window should also tend to increase and prolong hearing by bone conduction.

This theory, while not wholly convincing, is as satisfactory as any yet advanced so far as the writer knows. It does not, however, satisfactorily explain the increased hearing by bone conduction which occurs when the ear is closed by a finger placed lightly in the auditory meatus. This phenomenon, in the author's opinion, is better explained as follows: In hearing by bone conduction part of the force of the vibrations thus transmitted is expended upon the column of air in the auditory meatus and thus conveyed outward and dispersed through the medium of the surrounding atmosphere. When, however, a finger is inserted into the orifice of the meatus, this canal is converted into a closed cavity, and constitutes a resonance chamber in which the sonorous vibrations are collected and thrown back upon the drum membrane to augment those transmitted directly from the cranial bones to the annular ligament and foot-plate of the stapes. That the increased perception of sound is brought about by the walls of this artificially closed cavity acting as resonators, and not by compression of the contained air, is shown by the fact that the sound is heard loudest when the finger tip is placed lightly in the meatus, and is reduced when the finger is forced deeply into the canal so as to produce condensation of the inclosed air. The phenomenon in its causation is somewhat analogous to the increased noise which we have all experienced in a railroad train during its passage through a tunnel.

Leaving the question of its causation out of consideration, it is an established clinical fact that lesions of the conducting mechanism, not complicated by labyrinthine disease, are almost invariably accompanied by a prolonged period of hearing by bone conduction. The test for this phenomenon is as follows: Supposing the physician's ears and hearing to be normal, he holds the handle of a vibrating tuning-fork in contact with

his own mastoid process. As soon as he loses its sound the fork is transferred, without renewing its vibration, to the mastoid of the patient. If he then perceives its tone, it may be assumed that his bone conduction is abnormally prolonged. *Increased audition by bone conduction* is the second and, in the author's opinion, the most important functional sign of disease of the conducting apparatus.

Diminished Hearing by Bone Conduction.—That the influence of labyrinthine or auditory nerve lesions upon hearing by bone conduction should differ from that exerted by tympanic disease hardly requires explanation. The labyrinth and auditory nerve with the cortical brain centres constitute the perceptive mechanism proper. When the hearing is impaired as a result of disease in any of these structures, it follows logically that the hearing power is diminished by all the normal pathways of sound transmission. Hearing is, therefore, reduced by bone conduction and air conduction alike. The test for this functional change is exactly the reverse of that by which we detect increase in bone conduction. The vibrating tuning-fork is first held in contact with the patient's mastoid process corresponding to the ear to be examined. When he indicates that its tone is no longer heard, the fork is transferred to the mastoid of the physician, —provided that his ears are functionally normal. If its tone is now distinctly heard by him, the patient's hearing by bone conduction is assumed to be less than normal. *Loss or diminution of hearing by bone conduction* is characteristic of all forms of labyrinthine or auditory nerve disease.

We have, then, certain functional changes which show simply impairment of hearing, and which are met with in diseases affecting either the perceptive or the sound-conducting apparatus. Chief among these are (1) impaired audition for the watch or acoumeter, and (2) impaired hearing for the conversational voice and whisper. These changes do not materially aid us in locating the site of the lesion.

Of differential changes—*i.e.*, of functional changes characteristic of tympanic disease on the one hand, or of labyrinthine or nerve disease on the other—there are four which outrank all others in importance—viz.:

Functional changes characteristic of disease of the conducting apparatus: (1) Elevation of the lower tone limit, or loss of hearing for the lower tones of the musical scale. (2) Increased, or prolonged, audition by bone conduction.

Functional changes characteristic of labyrinthine disease: (1) Reduction of the upper tone limit, or loss of hearing for the higher musical tones. (2) Diminution or loss of hearing by bone conduction.

The writer believes that these changes in the tone limits and in the perception of sound as conveyed through the cranial bones, constitute the basic facts to be determined by our functional examination, and that the various differential tests are but convenient methods of demonstrating their presence or of determining their extent.

There are many functional irregularities shown by different individuals suffering from chronic deafness,—*e.g.*, disproportionate loss of hear-

ing for certain sounds, as of the conversational voice as compared with the watch and acoumeter, or *vice versa;* disproportionate impairment for certain words or consonants, etc. In spite of much careful study of such changes by O. Wolf, Bezold, and others, they have not yet been sufficiently worked out to be of great practical value, either in locating the lesion or as an indication of the line of treatment to be pursued. We shall now take up briefly the method of applying the various tests.

Method of Examination. — WATCH AND ACOUMETER. — In testing with a watch or acoumeter, the ear not under examination is closed by pressing a finger firmly into the orifice of the meatus. The watch is held for a moment close to the ear to be examined to familiarize it with the character of the sound. It is then removed four or five feet from the ear, and brought gradually nearer until its sound is heard, the distance in inches being noted. The experiment is now repeated once or twice for corroboration, the patient closing his eyes so that his imagination will not be stimulated by his knowledge of the position of the watch. The distance thus finally reached is carefully noted upon the test card.

It has been found that the watch-tick can be heard farther if the watch is first held close to the ear and the distance gradually increased than if the reverse method be employed,—*i.e.*, holding the watch outside of the hearing range and gradually approximating it to the ear.[2] The latter method is more reliable, since the imagination is brought less into play. If the watch sound can not be heard even when held within an inch of the ear, it should be placed in contact with the auricle, and the result, according to whether its sound can or can not be heard, should be thus noted upon the test card,—"heard only on contact," or "not heard on contact." In cases in which the watch is heard only on contact, or but a few inches from the ear, it is better to discard this test altogether and depend upon the acoumeter. One reason for this is the fact that such patients often make more or less pronounced functional gains for other sounds with relatively little change for the watch-tick; and again, the patient may so concentrate his attention upon his hearing for this particular sound as to become depressed or even neurasthenic because his functional gain as shown by this test does not seem sufficiently marked. With chronic aural disease as with other physical disorders it is desirable that the patient's attention should not be focused upon his symptoms. In cases of slight impairment of hearing, however, the watch-tick affords a most useful test.

The *acoumeter* is employed in exactly the same way as the watch except that one begins the test at a greater distance from the patient,—*i.e.*, standing at the opposite end of the room. The result is noted upon the test card either in inches or feet, according to the degree of deafness.

[2] This phenomenon is explained by the fact that the individual's power of auditory concentration and accommodation enables him to follow a sound once heard to a distance greater than that at which it is first perceived when the sounding body is gradually moved toward the ear.

WHISPERED AND CONVERSATIONAL SPEECH.—The patient's hearing power for whispered and spoken words or numbers furnishes us with a quick and convenient method of determining functional impairment or loss. We can not employ this test intelligently, however, without some preliminary study of *sound values,—i.e.*, of the varying carrying power of the different letters, alone and in combination.

Sound Values in Conversational Speech.—There are certain general facts which experience quickly impresses upon those coming in frequent contact with the partially deaf. We soon learn, for example, that the vowel sounds are heard farther and with greater distinctness than the consonants. A person with advanced catarrhal deafness may, therefore, hear the vowel sounds with comparative ease after the hearing for many of the consonants has become difficult, or possible only when they are spoken in loud tones. This fact explains the frequently repeated statement of the hard-of-hearing: "I hear your voice, but can not understand what you say." It also explains the not uncommon experience, in testing the hearing of such a patient for spoken words, that he repeats not the word we have employed, but one having the same vowel sounds: thus, we call the word "faster," and he says "master," guessing at the initial consonant. Further than this, we find that certain consonants are heard better than others; that in testing with whispered numbers, those beginning with s—*e.g.*, 67 or 76—may in some cases be heard easily at 20 or 30 feet, while numbers beginning with f or th—*e.g.*, 53 or 35—are heard only with difficulty at one-third of that distance. Or conditions may be reversed. Obviously the subject of sound values is one of considerable importance in practical otology.

Of the vowels a, e, and i are heard farther than o and u. While it has long been known that the vowels each possess a distinct tone or pitch (Helmholtz), it remained for Oskar Wolf[3] to prove that the consonants also possess each its own tone. By this is meant that each consonant, spoken without voice (*i.e.*, whispered) gives rise to a certain number of rhythmic vibrations per second, which determines its particular tone. This fact throws some light on the difficulties of the hard-of-hearing in interpreting conversational speech. For if the patient's ear has lost the power of responding readily to the tone inherent in a certain consonant, he will hear this consonant poorly or not at all. If in such a case the consonant in question is spoken in a tone of voice falling within the patient's range of tone perception, he will hear the voice but will not distinguish the consonant sound. Hence his confusion in following the trend of conversation.

The tones (pitch) inherent in the different letters are distributed rather widely over the musical scale. The letters l, m, n, and r are in the lower half of the musical scale, while the self-tones of the sibilants, s and sh, belong to the upper half of the scale. Bezold found that deaf-mutes who

[3] Sprache und Ohr, akustisch-physiologische und pathologische Studien, 1871.

possessed "islands" of tone perception which included the self-tones of the principal consonants, could be taught by means of the ear to speak; but that deaf-mutes having "islands" of tone perception which did not include the self-tones of the consonants, could not be taught through the ear.

The various elements of speech differ very considerably in the distances at which they can be heard by the normal ear. The s and sh sounds, for example, carry farthest and can be heard three times as far as f, v, t, th, d, and k. Again, f, v, t, d, and k are heard farther than b or p. While it is not important that the aurist should know the exact hearing distance for each consonant, it is quite essential that he should know and bear in mind that certain sounds are heard more easily than others. Thus, in the whisper test, the normal ear will be able to hear the words "sense" and "shine" farther than the words "pepper" and "five." When, however, the hearing is impaired by disease, the relative auditory acuteness for different sounds may be changed.

An approximate analysis of the relative loss of hearing for the different elements of speech may be made in the following way: Standing at a distance from the patient well beyond his range of hearing for the whisper, we begin our test with words or numbers spoken in a rather low whisper. It will be found that while most of the selected words are quite beyond his power of audition, certain words or numbers will be caught and repeated promptly and correctly. These words and the distance at which they are heard should be noted. We now move a few feet nearer the patient and repeat the test, when certain other sound values for this particular patient may be demonstrated, which also should be noted on the history card. The next step is to reduce the distance to a point where the patient can hear and repeat all words promptly and correctly. This distance must, of course, be recorded as representing his hearing distance for the moderately low whisper. We now possess rather definite data. Confining ourselves, for example, to the use of numbers, our results may read somewhat as follows:

RIGHT EAR:

Moderately low whisper,—6 feet (*i.e.*, distance at which all numbers are heard).

N-sounds,—*e.g.*, 19, 9, 99,—9 feet.

S-sounds,—*e.g.*, 16, 67, 76,—12 feet.

Such functional reactions are by no means uncommon in chronic aural disease. With such a record, one would not be likely to fall into exaggerated or erroneous conclusions as to a functional improvement when none had occurred. In other words, the haphazard use of words in testing is apt to lead to confusion, leaving the question of functional gain or loss in doubt.

So distinguished an authority as the late Professor Bezold[4] advised

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[4] Text-book of Otology, p. 72.

that we make use exclusively of numbers in our functional tests, expressing his belief that we can obtain with numbers all the data that might be gained by testing also with words. My own actual experience has led me to a quite different conclusion in regard to this particular point. If in testing, either with conversational voice or whisper, we employ only numbers, the patient quickly learns to associate certain sounds as the examiner utters them with the corresponding numbers, even though his actual hearing of them has not improved. His improvement is, therefore, more apparent than real. The writer believes that one should use words as well as numbers, the alternation of the one with the other furnishing the very best criterion of the patient's hearing power for conversational speech.

The *conversational voice* is also a useful means of determining the degree of impairment, but is of little value as a test for purposes of comparison. In cases of very advanced deafness, however, even the forced whisper may be unavailable, in which case it becomes necessary to use the voice, speaking in a very loud and moderately high tone. In such cases one must depend upon other tests to demonstrate slight changes either in the way of functional gain or loss.

Before leaving this subject, a word should be said as to the disparity between the estimated normal hearing distance for speech and whisper and the dimensions of the average testing room at the physician's command. We must remember that the sole purpose of functional testing is to determine the degree and character of the impairment. The aurist whose testing room is but 30 feet long soon learns to moderate the intensity of his whisper or voice in accordance with the space at his command, and his results are practically as reliable as they would be in a larger room or hall.

Determination of the Lower Tone Limit (Figs. 75 and 76).—For this purpose a set of tuning-forks is essential. Hartmann's set, consisting of five forks, supplies a convenient means of roughly estimating the lower tone limit in cases of very advanced catarrhal deafness. These forks represent C-tones one octave apart,—*i.e.*, 128, 256, 512, 1024, and 2048 double vibrations per second. But since the lowest fork of this series—*i.e.*, 128 d. v.—is about three octaves higher than the lowest tone perceived by the normal ear, a lower fork is required in cases of slight or moderate impairment of function. The large clamped fork (Fig. 76) is capable of executing either 26 or 64 double vibrations per second. With the clamps or weights attached to its prongs, it vibrates 26 times per second; and with the weights removed, 64 times per second. With this fork supplementing the Hartmann set, we can determine positively whether the lower tone limit is appreciably elevated. While 26 d. v. does not represent accurately the lower limit of normal tone perception, this fork is the lowest employed in many aural clinics, it being assumed that if the patient clearly perceives its tone, his hearing for the lower musical notes can not be greatly reduced.

In testing the patient's hearing for the lower musical tones, he should be required to close the ear not under examination. Using first the low,

clamped fork (26 d. v.), it is set in vigorous vibration and held near the external auditory meatus. If its tone is distinctly heard, it is assumed that his lower tone limit is normal, or nearly so; if heard but faintly and for a moment or two only, one may infer that the lower tone limit is elevated, and that the hearing for still lower tones—*e.g.*, 18, 20, 21, and 24 d. v.—is lost. If not heard at all, the clamps should be removed from the fork,

FIG. 75.—Hartmann's tuning-forks.

and the hearing tested for the next higher tone of this series of forks,—*i.e.*, C^{-1}, or 64 d.v. per second. If this also is inaudible to the patient, the hearing is tested with successively higher forks—*e.g.*, 128, 256, 512 d. v., etc.—until one is reached which the patient can distinctly hear. This tone, or vibration rate, is then recorded upon the test card as the lower tone limit, —*i.e.*, as nearly as it can be determined by this incomplete set of forks.

In using so large and heavy a fork as that producing 26 vibrations per second, the patient may feel the vibrations yet not hear its tone, and this

sensation he may mistake for tone perception. He must be made, therefore, to differentiate between the sensation due to the impact of the aerial movements upon the ear, and the perception of these movements as a continuous musical tone, or hum. If there be difficulty in making this difference clear to the patient, he may be required to describe the character of the sound, to state whether it is of high or low pitch, etc. If one ear only is involved, a comparison of the impression received by the opposite ear will usually remove any doubt as to whether the tone is heard by the diseased organ. Another possible source of error in the use of tuning-forks is the occurrence of overtones. In testing with unclamped forks,—*e.g.*, that producing 64 d. v.,—one usually hears immediately after it is set in vibration not only its fundamental tone (C^{-1}), but also certain higher tones, or harmonics, and these may be the only sounds heard by the patient. This, of course, might lead the examiner to very erroneous conclusions. One may easily eliminate overtones, however, by exerting momentary pressure with the thumb upon one of the prongs at a point as near as possible to the handle or shaft. It is important that this point be kept in mind.

With the limited set of forks above described, it is obviously impossible to determine the lower tone limit exactly. They enable us, however, in the great majority of cases to arrive at correct conclusions as to the character of the lesion so far as we are able to determine it from changes in the lower tone limit. For exact notation of the functional changes resulting from treatment, on the other hand, there is obvious advantage in the possession

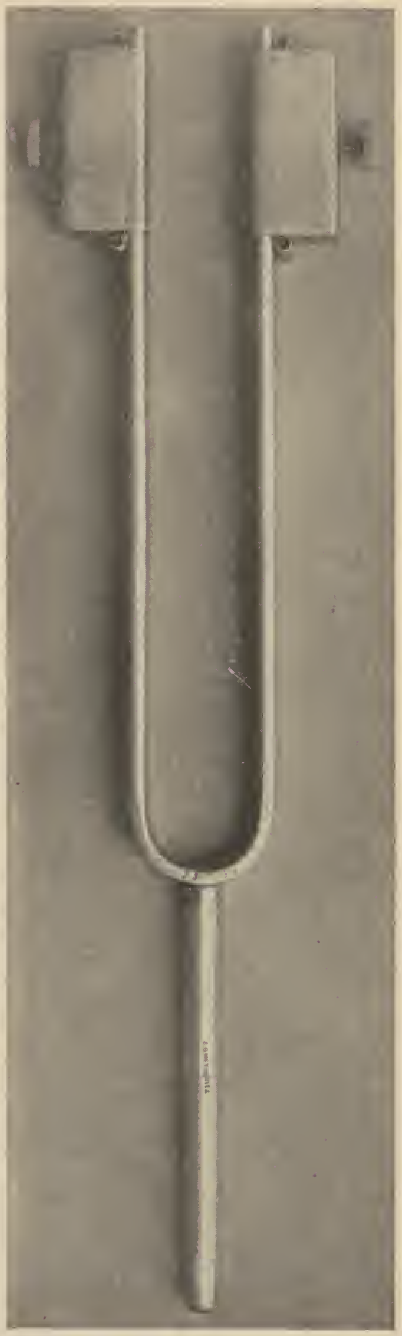
Fig. 76.—Large tuning-fork.

of a set of forks by which the lower tone limit may be exactly determined. For example, a patient may not be able to hear 128 d. v. (c), but may hear 256 d. v. (c′). Testing with the Hartmann set, we are obliged to note 256 d. v. as the lowest tone heard by the patient. But there are six full tones between 128 d. v. and 256 d. v. The lower tone limit may, therefore, be 144, 160, 171, 192, 213, 240, or 256 double vibrations per second. If now, after treatment, the patient can hear 128 d. v., it makes a great deal of difference whether his lower tone limit previously had been 144 d. v. (d) or 256 d. v. (c′), for in the one case he would have gained a single note, and in the other his tone range would have been extended by an entire octave.

Bezold's Tuning-forks.—Undoubtedly the most complete set of instruments for detecting gaps in the range of musical tone perception is that of Bezold and Edelmann of Munich. This set consists of ten large forks provided with clamps by which the tone of each fork may be varied, four smaller unclamped forks, two organ-pipes, and a modified Galton whistle. These forks are heavy, somewhat cumbersome, rather expensive, and are not made in this country. They can be ordered, however, through any reliable firm of instrument dealers, and enable one thoroughly to test the patient's hearing throughout the entire range of normal tone perception. They were designed especially for detecting tone gaps or islands of hearing in partial deaf-mutes, for which purpose they are undoubtedly the most complete and perfect instruments made. For measuring the degree of functional impairment in the hard-of-hearing, on the other hand, the full set is certainly not required.

For determining exactly the lower tone limit, the author in his private work makes use of a set of tuning-forks which were made for him according to his own specifications (Fig. 77). The prongs of these forks are of uniform width and thickness, the variations in pitch being regulated by gradations in their length. Without clamps they produce every full note from C^{-2} (32 d. v.) to c^2 (512 d. v.), and by weighting forks C^{-2}, D^{-2}, and E^{-2} notes corresponding to 16, 18, and 20 d.v. are obtained. These forks have proved most satisfactory and useful in the writer's practice.

Impaired hearing for the lower musical tones points to a tympanic lesion, and the loss of hearing for successively higher tones marks, as a rule, the progress of the disease.

Determination of the Upper Tone Limit (Fig. 78).—Diminution, or lowering, of the upper tone limit is most easily determined by the Galton whistle. This is practically a closed organ-pipe provided with an obturator, by moving which the tube may be lengthened or shortened at will. As the tube, or pipe, is shortened, the length of the sound wave is shortened and the vibration rate is increased. On the outer surface of the tube is a numbered scale,—0, 1, 2, 3, etc.,—provided with a mechanism by which changes in the length of the tube are indicated. By certain mathematical calculations these numbers and fractions thereof may be converted into the corresponding numbers of vibrations per second. This, however,

is not essential to practical diagnosis, the numbers themselves soon becoming associated in the aurist's mind with the normal in tone perception and certain degrees of departure therefrom. In testing with the Galton whistle, the instrument is held near the ear to be examined, the opposite ear being closed by a finger in the meatus. Air is propelled through the tube by compression of the rubber bulb attached to it. The patient will be likely to distinguish two sounds,—*i.e.*, a puffing or blowing sound as of escaping air, and a clear whistle. He should be required to indicate the point at which a clear whistle is heard. With the improved Bezold-Edelmann whistle, a clear note is heard with the marker at 0.5. Shortening the tube further than this, no clear whistle is heard. On the other hand, when the tube must be lengthened beyond 0.5 in order to obtain a note audible to the patient, it becomes evident that his upper range of tone perception is lowered. Some diminution of hearing for the extreme upper tones of the musical scale is physiological in old age, and may occur in certain disorders of the general nervous system. Generally speaking, however, any appreciable diminution of hearing for the upper musical tones in a person not over fifty years of age points either to a labyrinthine or nerve lesion, or to some aural disorder in which the labyrinth is secondarily involved.

An ingenious mechanism, which, if it could be made generally available, would add greatly to the accuracy and scientific value of our hearing tests, is the so-called tone-range audiometer devised by Dr. Lee Wallace Dean and Mr. C. C. Bunch, of the State University of Iowa.[5] Roughly speaking, this instrument consists of an electrically driven toothed wheel, the cogs of which come successively in contact with a magnet, the tone being produced by the rhythmic opening and breaking of the circuit. It is said to produce an approximately pure musical tone, the intensity of which can be controlled, and the pitch of which can be rapidly or slowly elevated or lowered between the tonal limits of 30 and 10,000 double vibrations. The advantages of such an instrument over tuning forks in the detection of gaps in tone perception are obvious. As with every notable advance in diagnostic method and accuracy, it is probable that this mechanism in the hands of its distinguished inventor will add to our scientific knowledge of disease by establishing shades of functional difference between certain closely related lesions,—shades of difference not definitely determinable by the older methods.

Changes in Hearing by Bone Conduction.—There are two methods of determining changes in the duration of hearing by bone conduction, one of which has already been mentioned. Assuming the examiner's ears to be functionally normal, the handle of a vibrating tuning-fork of 128 d.v. (c) is held in contact with his own mastoid process, and transferred as soon as its tone is no longer heard to that of the patient corresponding to the ear to be examined. If its tone is distinctly heard by him, it is inferred that bone conduction is increased; and if we count the number of seconds during which it is heard by the patient and find this to be, say, 10 seconds, we may record

[5] Dean and Bunch: Audiometer: Laryngoscope, August, 1919.

this result upon our test card as follows: "B. C., fork 128 d. v., increased (+ 10 seconds)." If the sound of the vibrating fork thus transferred from the physician's to the patient's mastoid is not heard by the latter, we may assume at least that bone conduction is not increased. The reverse experiment is now tried,—*i.e.*, the vibrating fork is held first against the patient's mastoid and transferred, when its tone becomes inaudible, to that of the examiner. If its tone is now audible to the physician, we know that the patient's hearing by bone conduction is diminished; and the degree of diminution will be determined by the length of time during which it is heard by the physician. Supposing this to be 10 seconds, the result may be noted thus: "B. C., fork 128 d. v., diminished (–10 seconds)." If the patient's duration of hearing is neither more nor less than that of the examiner, his bone conduction is charted "normal."

The method of examination just described is convenient and quite reliable so long as the examiner's ears, and particularly his hearing by bone conduction, conform to the normal standard. So soon, however, as his hearing becomes impaired, it is obvious that he will no longer be able to make correct deductions from a comparison of the patient's hearing with his own. If we wish to reduce this test to an absolutely correct basis, it is necessary that we should know the normal duration of hearing by bone conduction for certain forks, and determine whether the patient's hearing conforms to this standard. The corresponding forks of different sets vary, however, in the intensity of sound produced and in the period during which they can be heard. It is necessary, therefore, that the aurist should determine for himself the normal sound duration for his particular forks to be used in this test. This can be done with approximate accuracy by testing them (*i.e.*, determining in seconds their sound duration) upon a certain number of supposedly normal ears, which also show a normal standard as gauged by other tests,—*e.g.*, watch, acoumeter, whisper, Rinné, etc. And as two forks—*i.e.*, the 128 d. v. and 256 d. v. forks of the Hartmann set—will suffice for all tests of bone conduction, it is not a difficult matter to determine their normal period of sound transmission through the cranial bones. In testing by this method, the fork should be struck forcibly, to obtain its maximum vibration, and the handle at once placed in contact with the mastoid process of the ear under examination. The number of seconds during which its sound is heard by the patient should be counted by means of a stop watch. Supposing, for example, that we are using a fork the normal duration of which has been found to be 20 seconds, it is an easy matter to determine by how many seconds the patient's hearing by bone conduction is either increased or diminished. This method of testing bone conduction is given as the only one open to the physician who is himself the victim of advanced aural disease. With fairly normal ears, however, the first method is much quicker, infinitely more convenient, and yields sufficiently accurate results.

Impairment or absolute loss of hearing by bone conduction in one ear is sometimes difficult to determine, from the fact that the sound may be transmitted through the cranial bones to the opposite ear. Usually, however,

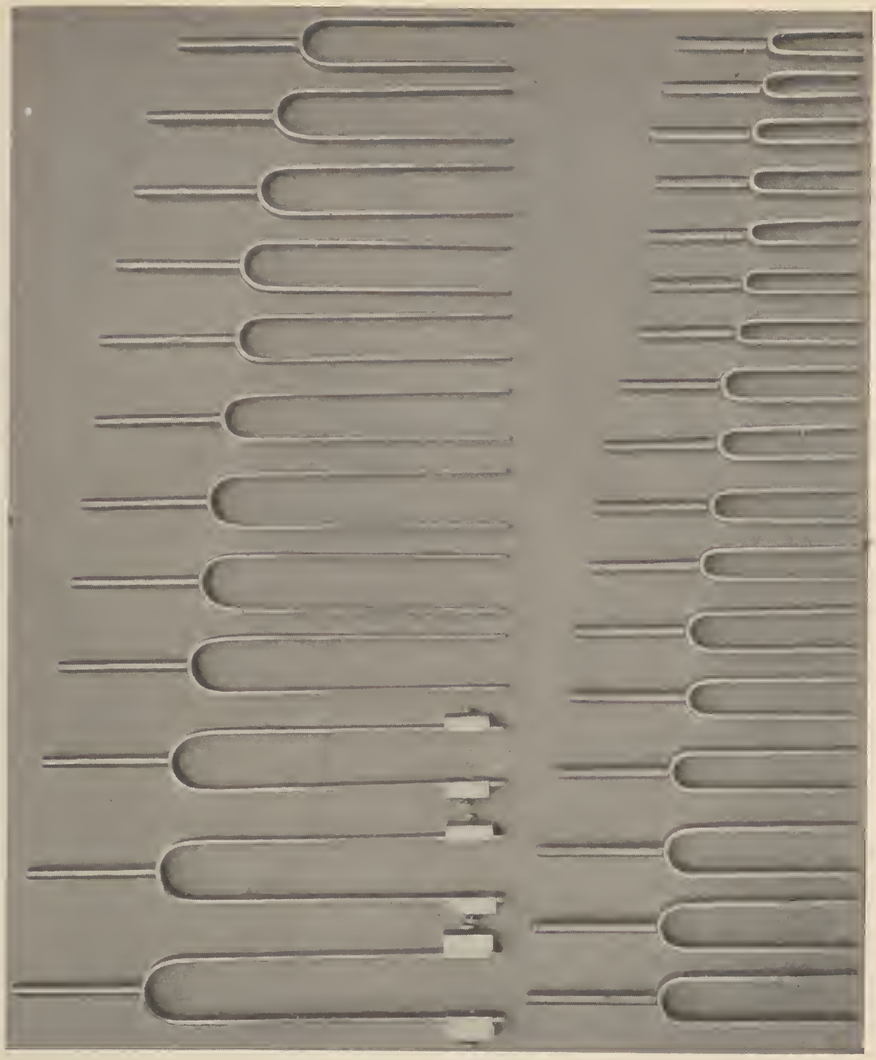

Fig. 77.—Kerrison's set of tuning-forks.

The number opposite each fork corresponds to the number of double vibrations which that particular fork (in producing its fundamental tone) executes. The number opposite forks C^{-2}, D^{-2}, and E^{-2} indicate the number of double vibrations which these forks execute when the clamps are removed. With the clamps adjusted and moved to different positions upon the prongs, much lower tones—*i.e.*, tones corresponding to 14, 16, 18, 20, 22, 24, and 26 double vibrations per second—can be produced

Fig. 78.—Galton whistle.

one can obtain sufficiently positive data to enable one to draw correct conclusions as to the character of the lesion.

Having tested the patient's hearing both as to the tone limits and as to changes in bone conduction, we are in a position to form a fairly definite opinion as to the site of the lesion. In the writer's opinion, further tests, while of undoubted value, are for the most part corroborative. That is to say, with elevation of the lower tone limit and increased bone conduction, the hearing for the upper tones remaining normal, the inference is definitely in favor of disease confined to the conducting apparatus. If, on the other hand, the upper tone limit is lowered, the lower limit but little changed, and hearing by bone conduction is decidedly diminished, we are not likely to prove in error in concluding that the labyrinth or nerve is chiefly at fault.

Schwabach's Test.—Schwabach attempted to determine the periods in seconds during which tuning-forks of different pitch are heard through the cranial bones by the normal ear. He proved that there is an absolute increase in bone conduction in diseases of the conducting apparatus. His test, therefore, proposed the determination of the patient's duration of hearing by bone conduction of a large number of forks, and their comparison with the normal standards therefor. This test adds little to the knowledge obtained from the simpler experiment by which increase or diminution of bone conduction is determined. Its results are corroborative.

Rinné's Test.—Rinné's experiments proved two things,—viz.: (1) that the normal ear hears a vibrating tuning-fork twice as long by air conduction as through the cranial bones, and (2) that in tympanic disease this ratio between air conduction and bone conduction may be absolutely reversed. The c fork (128 d. v.) and the c′ fork (256 d. v.) of the Hartmann set are appropriate for this test. For young adults the c′ fork is preferable, but with patients past middle life, in whom hearing by bone conduction is physiologically reduced, the c fork (128 d. v.) gives more reliable data. Rinné's test is applied in the following way: the fork is set in vigorous (maximum) vibration and the handle at once placed in contact with the mastoid corresponding to the ear to be examined. As soon as the patient indicates that its tone is no longer heard, the fork is removed from contact with the cranium and the ends of the still vibrating prongs are brought near to and opposite the orifice of the meatus. If now he again hears its tone and continues to hear it during a period approximately equal to its duration by bone conduction, we must regard this as the normal ratio between hearing by bone conduction and by air conduction. This in otological literature is spoken of as a "*positive Rinné.*" It may be found, however, that after hearing by b. c. ceases, the patient will no longer be able to hear by a. c. This, of course, points to disease, or at least derangement, of the conducting apparatus. We must now reverse the experiment, holding the vibrating fork first in front of the ear, and transferring it when its sound is no longer heard to contact with the mastoid. It is possible that its tone will again be heard through the cranial bones during a period about equal to its previous duration by air conduction. This reversed ratio indicates advanced tympanic disease, and is spoken of

as a "*negative Rinné.*" This is one of the most reliable and useful tests of chronic middle-ear disease. No other functional test shows so graphically the various stages of functional impairment. Thus, in the earliest stages of tympanic disease the hearing by a. c. will be very slightly diminished while the hearing by b. c. will be very slightly increased. The ratio between b. c. and a. c. will, therefore, not be reversed, the period of hearing by a. c. after hearing by b. c. has ceased being simply shortened. This is spoken of as a "*diminished positive Rinné.*"

The diagnostic significance of the various reactions to this test in unilateral aural disease may be stated as follows:

A. Hearing noticeably impaired, Rinné negative, the ratio between b. c. and a. c. being reversed; these reactions would point to an advanced lesion of the conducting apparatus.

B. Hearing noticeably impaired, Rinné positive, the ratio between b.c. and a. c. being normal, would suggest disease of the labyrinth or auditory nerve.

C. Hearing not appreciably impaired, Rinné positive and showing normal ratio, would suggest a normal ear.

D. With hearing but slightly impaired, a "diminished positive Rinné" would indicate a comparatively early stage of tympanic disease. In such a case the progress of the lesion is likely to be marked by progressive shortening of the period during which hearing by a. c. outlasts hearing by b. c.

The prognostic value of this test is indicated by the following fact, as to the correctness of which I am sure that all experienced aurists will bear me out,—viz., that when, as a result of chronic or slowly developing tympanic disease, the ratio between b. c. and a. c. is absolutely reversed, the hearing may be improved by treatment, but can never be restored to the normal standard.

Weber's Test.—This consists in holding the handle of a vibrating tuning-fork (128 d. v. or 256 d. v.) in contact with the median anteroposterior line of the vertex of the skull. The sound will be transmitted through the cranial bones, and, since bone conduction is increased in tympanic disease, the sound will be conducted in greater volume to the diseased ear in all unilateral lesions of the conducting mechanism. The patient, therefore, has the impression of hearing it altogether with the diseased ear. On the other hand, if hearing by bone conduction is diminished in the diseased ear, as is the case in lesions of the labyrinth or auditory nerve, the sound will be appreciated chiefly by the normal ear. In unilateral deafness, therefore, the sound of a tuning-fork held in the mid-line of the skull will be referred by the patient to the diseased ear if the lesion is located in any part of the conducting apparatus, and to the sound ear if the disease is confined to the labyrinth or auditory nerve. It is a recognized fact that in certain individuals with perfectly normal ears the hearing by bone conduction may be referred chiefly to one side or the other, purely as a result of inequalities in the thickness, or other anatomical differences, between the opposite sides of the skull. This, however, is exceptional. In the average run of cases Weber's test is an easily applied and useful corroborative test, confirming what we have

already learned as to the relative increase or diminution of bone conduction in one ear as compared with the other.

Gelle's Test.—This test is dependent upon the fact that any force exerting sudden inward pressure upon the stapes pushes this ossicle farther into the oval window and, by increasing intralabyrinthine pressure, reduces tone perception, whether the sound wave has reached the tympanum by air conduction or by bone conduction. Gelle's experiment consists of condensing the air in the external auditory meatus by means of a Siegel's speculum and bulb, or similar apparatus, a vibrating tuning-fork being held, meanwhile, in contact with the skull. Normally, as the drum membrane and ossicles are forced inward, intralabyrinthine pressure is increased and the sound of the tuning-fork is thereby diminished. As the pressure upon the drum membrane is removed, the stapes moves outward, intralabyrinthine pressure regains its equilibrium, and the sound of the vibrating fork increases in volume. In this way the intensity of the sound may be alternately increased and diminished. It is held that when tympanic changes causing fixation of the ossicles have occurred, the labyrinth will not be affected by condensation of air in the external auditory meatus, and hearing by bone conduction will not be diminished. A negative result is, therefore, held to point to ossicular fixation. So far as the writer can see, this test is of theoretic value in only one condition,—viz., when the stapes is firmly fixated or immobilized, within the oval window. It is not of great practical value in diagnosis.

Bing's Test.—This experiment is mentioned in most text-books as a means of differentiating labyrinthine from tympanic lesions. A vibrating tuning-fork is held in contact with the skull,—preferably upon the mastoid process of the ear to be tested. As soon as its tone can no longer be heard, a finger is introduced into the external auditory meatus. It is claimed that the tone will again be distinctly heard if the disease be located within the labyrinth, but will not be heard, or will be heard but faintly and during a diminished period, if the lesion be confined to the tympanum. This test is somewhat unreliable from the fact that in the early stages of either labyrinthine or tympanic disease the sound will recur after closure of the meatus, and in very advanced stages the experiment is likely to give negative results whether the disease be located within the labyrinth or the middle ear. With marked increase in bone conduction, one is not likely to think of labyrinthine disease because the sound of a vibrating fork held against the skull can be prolonged by closing the meatus, nor will one doubt labyrinthine disorder in the presence of marked diminution of bone conduction because this experiment results negatively.

Total Deafness.—In the foregoing pages we have described the various hearing tests which are of use in the analysis of partial deafness,—*i.e.*, the impairment of such patients as are commonly spoken of as "hard of hearing." Practically all cases of functional impairment due to tympanic disease, and many cases in which the perceptive mechanism is also involved, come under this classification. There is, however, another class

of cases in which many of these tests are of little or no value,—I refer to cases of bilateral or unilateral impairment so great as to approach or actually to constitute total deafness. When one ear is very nearly or totally deaf and the other ear retains in large degree its quota of hearing power, the possibilities of error in diagnosis are not inconsiderable. Cases have come under the writer's observation in which the patient has been subjected to much useless treatment simply because a careless or inexperienced practitioner had failed to recognize the fact that one or the other ear was totally or hopelessly deaf. Such profound deafness always means disease of the cochlea or of the auditory nerve. A word must therefore be said as to the determination of profound or total deafness.

Total bilateral deafness is not difficult to determine. In the first case, the patient is unable to hear a word that is said, even though it be spoken in a loud voice or shouted close to the ear. With tuning-forks also, unless there be remaining islands of tone perception, he hears no sound. It sometimes occurs with tuning-fork tests that the patient receives the impression that he hears the sound when in reality he only feels the impact of the aerial vibrations against the skull. This is particularly apt to occur when we use a large fork in maximum vibration. We are usually able to detect this error by his inability to describe the sound correctly.

With total deafness, bone conduction also is lost. But here again we must guard against apparent contradiction resulting from the patient's inability to differentiate between his sense of the vibrations against the skull and tone perception.

Absolute Deafness in One Ear.—This, when the opposite ear is functionally normal or nearly so, may be difficult to determine. The tuning-fork tests of tone perception, for example, present the following difficulties. With the normal ear closed by a finger in the meatus, the patient can not hear the lower musical tones,—*i.e.*, tones from 18 to 42 d. v. But when we reach 64 d. v., or a little higher, it will be found impossible to exclude its tone from the normal ear, however tightly it be closed. If now he be directed to close both ears, and still can hear the tuning-fork and with equal intensity, we may conclude that it is heard only by the sound ear.

Loss of hearing by bone conduction may be even more difficult to determine from the fact that tones of a vibrating tuning-fork held at any point on the skull may be transmitted through the cranial bones to the sound ear. It is best to begin by holding the fork in contact with the mid-line of the skull (Weber). The patient usually states that the sound is heard altogether by the normal ear. The experiment is now several times repeated, selecting each time a point nearer the diseased ear. If finally, with the tuning-fork in contact with the mastoid of the diseased ear, the patient still refers its sound to the healthy organ, we may be sure that hearing by bone conduction is completely lost in the ear under examination. Unfortunately, the value of this test may be negatived by the patient's inability to interpret correctly his own impressions.

With many of these cases the final test of profound or total deafness is

the patient's inability to hear the conversational voice. Voice sounds may, however, be heard by the sound organ, even though it be closed by a finger tightly pressed into the meatus. To meet this difficulty we have a reliable instrument in Barany's noise apparatus (Fig. 79). This consists essentially of a small metal box provided with an ear-piece and containing clock-like machinery which when wound up produces a continuous rough metallic sound. It is used as follows: The ear-piece is introduced into the canal of the sound ear and by pressure upon a button the machinery is set in motion. While this sound is being poured into the normal ear, words or numbers are spoken in a loud voice close to the diseased ear. If they are not heard, we may be sure that the ear under examination is totally deaf. If the words are heard, we may be equally confident that they are not heard by the ear into which the noise apparatus is pouring its sound, and that the diseased ear is, therefore, not totally deaf.

While the profound type of deafness above described is never met with in lesions confined to the conducting apparatus, it is the rule in certain lesions of the inner ear,—*e.g.*, diffuse suppurative labyrinthitis. In such cases Barany's noise apparatus, or some similar instrument, is absolutely essential.

Fig. 79.—Barany's noise apparatus.

Malingering; Simulated Deafness.—Pretended deafness is said to be comparatively common in countries where army service is compulsory. In America it is met with chiefly in the case of impostors seeking indemnity on account of pretended injuries to one or both ears.

Simulated bilateral deafness, if the individual persistently refuses to give evidence of any hearing power, is naturally difficult to detect. To disprove complete bilateral deafness, one must depend on one's ability to catch the individual off his guard. It has been suggested that by making disparaging remarks about him in the presence of a third party one may be able to determine by changes in his facial expression his ability to hear the conversational voice. Usually, however, the pretense of complete bilateral deafness is too difficult to maintain, and as a rule it is unilateral deafness the genuineness of which the aurist is called upon to determine.

There is no absolutely reliable test for feigned deafness, the astuteness of the malingerer and the cleverness and knowledge of the physician being elements at play in every case. The following are among the tests employed.

1. Assuming in a given case the lesion to be confined to the sound-conducting mechanism, a vibrating tuning-fork is held to the mid-line of the skull (Weber). Its sound should be referred to the side of the lesion; the malingerer naturally states that it is heard by the sound ear.

2. Testing the patient's hearing for whispered speech through a tube dividing

terminally into two smaller tubes—one for each ear—he is asked in which ear the words are heard. If the terminal tube communicating with the sound ear be blocked or closed, and he claims that the words are heard only by the presumably sound ear, it is assumed that the deafness in the opposite organ is not real.

3. When only partial deafness is claimed, the various tests—*e.g.*, as to hearing distances, tone limits, etc.—are made. Repeating these tests, the malingerer's memory usually fails him, and his conflicting statements are likely to show that his answers are not to be relied upon.

4. The individual having been blindfolded, the examiner stands behind him, and alternately tests the two ears rapidly with various forks. This frequently confuses the malingerer and brings answers so conflicting and contradictory as to demonstrate his lack of good faith.

5. A device which the writer recently hit upon was conclusive so far as the case in question was concerned. The patient had received an injury in the course of his work for a large commercial company, and claimed to have become deaf in his right ear. For this he proposed to sue the company. The drum membrane appeared normal. His answers to the usual tests showed a degree of deafness which could only have resulted from a labyrinth injury. Arguing that such a disturbance of the cochlea would be likely to involve also the vestibular mechanism, the writer made a caloric test of the presumably deaf ear. This resulted in a quick and normal reaction, and during the confusion incident to the induced vertigo, it was easy to determine that his apparent unilateral deafness had been assumed.

Since the above was written, American aurists have acquired much practical knowledge in the application of these tests in the examination of men striving to evade military service on account of pretended deafness. It may, therefore, be worth while to state briefly the tests which the writer, as a member of an advisory examining board, found most useful, and to describe the routine method of applying them.[6]

In the first place, let me emphasize the fact that it is important in dealing with a suspected malingerer that he shall receive no slightest hint that he is under suspicion. The more unsuspecting and credulous the examiner may appear, the more readily and conclusively will the suspect respond to the tests. The most glaring evidences of deception should therefore pass without comment or apparent notice until the examination is completed.

Most malingerers of deafness who are subject to the military draft know that deafness of a certain grade in either ear will exempt them from service. Complete bilateral deafness is, therefore, for various and obvious reasons rarely, if ever, claimed.

Two types of unilateral deafness are assumed, *i.e.*, (1) deafness advanced but not complete; and (2) absolute deafness.

My own routine method of examining these cases is about as follows: Only one man at a time is admitted to the examining room. I learn from him which is the deaf or deafer ear, and whether he regards this deafness as partial or complete. If only one ear is complained of, I make a rapid test of the better ear to determine that it is functionally sound or at least only slightly subnormal. This preliminary testing of the sound ear is essential to a proper interpretation of the tests to be applied later.

Weber's Test.—I now apply a vibrating tuning fork of 256 double vibrations to the vertex of his skull, and if he refers the sound to his supposedly

[6] The following pages are taken almost verbatim from a report by the writer made before the American Otological Society in June, 1918.

deaf ear, I am favorably impressed as to his honesty. If he refers it to his sound ear, I become skeptical, and proceed to the next test.

Loud Voice Test.—The suspect's eyes are now blind-folded. Requesting him to close with a finger his better ear, which has already been determined to be approximately sound functionally, I repeat words and numbers to him at first in a low voice and then in progressively higher and louder tones. If, when I have reached a pitch and intensity at which he should be able to hear and interpret the words with his sound ear even though tightly occluded, he still states that he cannot hear them, I know at least that he is an intentional malingerer. This is a useful test which will expose many impostors. An alert individual, however, may throw this procedure out of court by stating that he hears the sound through his occluded normal ear.

Stethoscope Test.—For this the ordinary clinical stethoscope with funnel-shaped chest piece is used. One ear piece is completely occluded with wax. Occlusion with cotton will not exclude the sound. Standing behind the examined, the stethoscope is adjusted with the occluded ear piece in his "deaf" ear. Words in a low voice are spoken into the funnel-shaped chest piece, which naturally he should hear perfectly. The stethoscope is removed for the ostensible purpose of trying some other test, and then replaced, the occluded ear piece being this time placed in his sound ear. If he is able to hear now approximately as well as before, we have fairly sound evidence that his deafness is either assumed or grossly exaggerated. This is a fairly reliable test.

Tests for Eliciting Contradictory Responses.—The eyes are now uncovered, the sound or better ear is closed with a finger, and the "deaf" ear is rapidly subjected to the commoner classical tests, *e.g.*, hearing distance for watch, acoumeter, whisper or conversation voice; tuning fork tests to determine lower tone limit, etc. His responses, so far as he admits hearing in any of these tests, are carefully noted. Following this, he is again blindfolded and the same tests are repeated many times, fairly rapidly and in varying order. If he is a malingerer and has not in the first instance claimed almost total deafness, his responses will almost surely demonstrate incongruous and contradictory variations.

Lombard's Test.—This test, which requires a Barany noise apparatus, is one of the most dependable means of determining absolute or very advanced deafness confined to one ear. It depends upon the fact that to the normal man the sound of his own voice is necessary to the regulation of its tone and intensity. It is carried out as follows: the noise apparatus is adjusted to his sound ear and its machinery started in order to accustom him to its grating noise. He is given a book and told to read aloud in his natural voice and not to stop reading when the noise instrument is set in action. As soon as the noise begins, a man whose opposite, or open, ear is profoundly deaf will at once raise his voice, and if his deafness is absolute he may literally shout. The malingerer, on the other hand, claiming a one-sided deafness which is not real, will continue to read in an even tone or in a tone only slightly elevated. This is a test which a malingerer who has been

coached may easily turn to his advantage. Otherwise it is one of the most useful at our disposal.

Cochleo-palpebral Test (*Gault*).—This test depends on the fact that if a sudden and unexpected noise is produced near either ear, there occurs a slight winking movement, or contraction of the lids, in the corresponding eye. If, therefore, in a case of supposedly unilateral deafness, the sound ear is tightly closed and a slight noise, produced near the ear under suspicion, is followed by a contraction of the lids, however slight, on the same side, this is accepted as evidence of the sound having been heard in the ear in which deafness is claimed. It is clear that this test might be even more valuable in the case of a clever malingerer claiming bilateral deafness, when a noticeable palpebral contraction in response to a slight noise might constitute the only proof of the unreality of the auditory defect.

Bilateral Deafness.—Naturally, as there are many cases representing all grades of bilateral deafness, malingering may take the form merely of an exaggeration of an existing defect. If such a person were persistently to assume absolute or very profound bilateral deafness, it is difficult to say what tests would expose the deception. The time-honored tricks of attempting to ensnare him by a sudden and startling question, the use of insulting or disparaging remarks, etc., may be of value in certain cases but are certainly not to be relied upon. The majority of malingerers, however, do not pretend to complete deafness. This is a fortunate fact for the reason that partial though disabling deafness burdens the malingerer with the necessity of maintaining uniformity in his responses to the various tests. The surest means of exposure in such cases is the application of the various classical tests first to one ear and then to the other, and then, after blind-folding him, repeating the same tests and comparing the two sets of responses. In my opinion, the most useful single test is the repeated determination, using a full set of tuning forks, of what he claims to be the lower limit of tone perception in each ear. In such repeated tests, a man would have to possess both a phenomenal memory and a wonderfully trained musical ear to avoid conflicting replies.

CHAPTER IV.

DISEASES OF THE EXTERNAL EAR (AURICLE; EXTERNAL AUDITORY MEATUS).

INFLAMMATORY DISEASES OF THE AURICLE.

Traumatic Auricular Dermatitis.—This condition is occasionally seen as a result of the slighter injuries to the ear,—*i.e.*, injuries resulting in contusion or abrasion of the cuticle, but not seriously involving the perichondrium. It begins as a simple erythema, is usually accompanied by some degree of pain and sensitiveness to touch or pressure, and soon spreads more or less widely over the auricle. To establish its traumatic origin, it must be traceable to an abrasion or injury of the skin at the orifice of the meatus or at some point on the pinna. It may run its course as a simple erythema or, developing localized points of infection, become pustular in character. Such a lesion, unless too long neglected, is usually quite responsive to treatment.

The TREATMENT should begin with thorough cleansing of the part. If for this purpose an antiseptic drug is required, a solution of carbolic acid, 1 to 200 parts, is preferable to bichloride of mercury, which in itself is too irritating to the skin. After cleansing, the auricle should be thoroughly dried, and an antiphlogistic ointment or lotion applied, according to the severity of the inflammation. If of very acute type,—*i.e.*, of sufficient severity to cause considerable pain or discomfort,—it is well at the beginning of treatment to protect the ear by means of a large dressing of sterile gauze. The ointment or lotion may be applied upon a thin layer of gauze, and a dry gauze dressing applied over this. It may be said, however, that in many cases dermatitis of this type will make a perfectly satisfactory recovery without other treatment than cleansing of the part and protection from fresh injury.

Erysipelas of the Auricle.—This disease may be secondary to the traumatic lesion above described, or it may have its origin in some slight injury or abrasion of the auricle or auditory meatus. Erysipelas of the auricle does not differ materially from the same lesion in other parts of the body. It rarely remains localized in the auricle, usually spreading to some extent to the face and scalp, so that it is apt later to assume the characteristic features of facial erysipelas. The redness, swelling, and tenseness of the skin are much more marked than in the simple traumatic type of erythema, and the line of demarcation between the inflamed and normal areas presents a greater contrast. The pain is usually intense. In addition to the local manifestations, there are usually present fever, accelerated pulse, and other symptoms of systemic derangement.

The TREATMENT is that of facial erysipelas: Rest in bed, catharsis,

tonics, local cleansing, application of antiseptic or antiphlogistic ointments or lotions. Many of the local measures advocated have seemed to the author to exert little or no influence on the course of the lesion, and, though recovery is the rule, one is not always sure to what extent this happy result is due to the remedies applied. The writer is inclined to believe that for severe cases the use of a leucocyte extract (Hiss) will prove to be the treatment insuring the most definitely favorable results. In a later chapter dealing with serum therapy in the treatment of aural disease, the control of erysipelas by the Hiss leucocyte extract will be briefly discussed.

Frost-bite.—The auricle from its exposed position is peculiarly vulnerable to this accident. The symptoms are characteristic, but are often at the time overlooked by the sufferer. The ear, which has been tingling with cold, is suddenly found to be insensitive to touch. On examining the ear, the affected part of the auricle—oftenest the tip of the lobe or margin of the helix, or both—is found to be dead white and absolutely insensitive. The adjoining uninvolved part of the auricle is deep pink or red. Later—unless the circulation is quickly re-established—the frozen part becomes purplish, and still later may become dark brown or black, the part so discolored being afterward separated as a slough. This necrosis may not involve the cartilage, but simply the superficial tissues. In still milder cases distinct sloughing does not take place, the parts gradually regaining tone and, after desquamation of the skin has taken place, returning to an apparently normal condition. That the tissues do not quickly return to an absolutely normal state is shown, however, by the fact that even in very mild cases the parts attacked are usually for a considerable period thereafter particularly vulnerable to the influence of cold, and therefore to recurrence on comparatively slight exposure.

Treatment.—The first indication is to re-establish the local circulation in the part frozen. Heat should not be applied, nor should the sufferer be taken into a warm house or room. If snow is on the ground, vigorous rubbing with snow, or better still with the hands after they have been made cold by contact with the snow, is a time-honored custom, and probably the best method which can be employed. Restoration of the local circulation will be announced by the return of local sensation and pain. Later the ears must be carefully protected from renewed exposure, and, if it becomes evident that sloughing is to some extent inevitable, they should receive antiseptic care and treatment in accordance with the surgical laws governing the management of necrotic or gangrenous wounds.

Auricular Eczema.—Clinically, eczema of the external ear is recognized in two clearly differentiated forms, the acute and the chronic.

Etiology.—The acute form occurs far more frequently in childhood than in adult life, and in the uncared-for and ill-fed children of the tenements than among those living under more favorable hygienic conditions. Early childhood and depressed constitutional states must, therefore, be regarded as strongly predisposing factors. Digestive disturbances, and especially intestinal disorders, also seem to influence the development of

the disease. The chronic form is seen oftener in adult life and old age, and with particular frequency in those in whom the rheumatic or gouty diathesis is present.

Symptoms of Acute Auricular Eczema.—Three stages of the lesion are frequently seen in aural clinics: (1) The erythematous stage, in which the skin is intact, but red, somewhat swollen and angry looking. (2) The vesicular stage, in which the skin is more or less covered with vesicles. From some of these serum exudes which bathes the inflamed surface, finally drying in the form of scales. (3) A stage in which the area involved is completely denuded of its epithelium and presents a raw and bleeding surface.

The chief symptom is that of the very distressing local irritation,—*i.e.* the intolerable itching. This is present in all stages. In the erythematous stage there is sometimes in children very considerable pain, giving rise in some cases to moderate elevation of temperature.

The parts most frequently involved are (1) the postauricular sulcus and contiguous parts behind the ear; and (2) the concha and often the outer half of the membrano-cartilaginous meatus. Children find it impossible to resist the inclination to scratch the intensely itching surfaces, and this often adds a superficial infection to the primary lesion.

Treatment.—Fortunately, these cases usually respond readily to proper management. The writer usually begins with internal remedies to cleanse the intestinal tract and regulate the digestion. For this purpose the following well-known formula for rhubarb and soda gives very satisfactory results:

℞ Extract. rhei fluid.,
Sodii bicarbonatis,
Spirit. menth. piper., āā ʒi;
Aquæ dest., q. s. ad ℥iv.
M. Sig.—One teaspoonful in wineglass of water t.i.d., a.c.

The local treatment calls for cleanliness, application of an astringent ointment, and protection from mechanical irritation (scratching, etc.). The parts should be gently bathed with warm water and castile soap, and thoroughly dried. They should then be covered rather thickly with some soothing and astringent ointment. The parents should be instructed to reapply this ointment two or three times daily as may become necessary, and not again to bathe or apply water to the part until so instructed. The ointment which the writer is accustomed to prescribe is as follows:

℞ Unguent. zinci oxid.,
Petrolati, āā ℥ss.
M. et ft. unguentum.

When the skin is very acutely inflamed, much quicker and more satisfactory results are obtained by applying the ointment once daily and protecting the parts in the interim by means of a dressing of sterile gauze. It is surprising how rapidly and completely most cases of acute auricular eczema will clear up under this treatment if faithfully carried out.

A condition the treatment of which presents some difficulties is found in cases in which acute eczema of the meatus and auricle coexist with discharge from the middle ear. In such cases the syringing employed to cleanse the canal of pus exerts an unfavorable influence upon the eczema. Under such circumstances the eczematous surfaces should be guarded from the irrigation fluid by thick layers of the protecting ointment. When the aural discharge is not profuse, manual cleansing and drying of the meatus, followed by the introduction of gauze wicks,—the whole ear being covered with a gauze dressing,—may with advantage be substituted for irrigation of the ear. Such a dressing would have to be renewed daily.

Chronic eczema of the auricle usually involves also the outer half or third of the external auditory meatus. Two clearly distinguishable types are recognized: (1) The squamous variety, in which the outer part of the meatus and part of the concha are covered with dry, thin scales of exfoliated epithelium. Removal of these scales exposes a sensitive and easily irritated surface. (2) The so-called sclerotic type of eczema, in which the skin lining the outer part of the meatus and covering parts of the auricle is greatly thickened. The calibre of the meatus is in some cases greatly reduced by superposed layers of exfoliated epithelium, removal of which is apt to leave an excoriated and bleeding surface. In both types of chronic eczema, itching is usually a more or less prominent symptom, and the patient's effort to relieve this by scratching tends to prolong and exaggerate the condition.

TREATMENT.—Tonics and—in cases in which such a diathesis exists—antirheumatic remedies seem to influence some cases favorably. The local treatment calls for the removal of the scales, or exfoliated epithelial layers, lining the canal and concha. Unfortunately, this is often quite difficult or impossible without leaving a raw or abraded surface, which opens the door either to infection or to the formation of scabs or crusts, beneath which epithelial hyperplasia and exfoliation proceed as before. It is well, therefore, in some cases to apply an emollient ointment for several days before attempting to remove the thickened and adherent surface layer. For this purpose the following is quite satisfactory:

℞ Glycerini, ʒi;
Lanolini, ʒiii;
Unguenti petrolati, ℥i.
M. et ft. unguentum.

This should be applied thickly each day until the abnormal surface covering of the canal is softened, and can be easily removed without injury to the underlying skin. The parts should then be thoroughly cleansed with 95 per cent. alcohol, and covered by some protective ointment,—*e.g.*, the zinc oxide ointment, or the following:

℞ Ichthyoli, ʒi;
Acidi salicylici pulv., gr. x;
Zinci oxidi, ʒss;
Unguent. aquæ rosæ, ℥i.

Later, occasional swabbing of the ear with alcohol helps to restore to the skin its normal tone. It must, however, be kept soft by frequent applications of some astringent ointment to prevent re-formation of the scales. The condition regularly improves under careful local treatment, but I know of nothing which will absolutely insure against recurrence after treatment has been discontinued.

Auricular Perichondritis.—This is an inflammation involving the auricular perichondrium, and usually resulting in an effusion of pus or serum between it and the auricular cartilage. It is said in some cases to develop idiopathically, or at least without known cause (Politzer); but in the vast majority of cases it is traceable to a direct infection, spreading either from a neglected furuncle of the cartilaginous meatus, from a lacerated wound of the auricle, or the infection may be traced to a plastic operation involving the cartilage,—*e.g.*, the plastic work upon the cartilaginous meatus forming part of the "radical operation."

Symptoms.—The onset is announced by gradually increasing auricular pain, which soon becomes exceedingly severe and usually interferes with sleep. Upon examination we find at the beginning a small area of localized swelling. The area involved is dark red or purplish red, is hard, tense, and unyielding to the palpating finger, and is exquisitely sensitive to manipulation or pressure. These changes may at first be confined to a very small area, from which they may spread gradually in all directions over the anterior surface to the extreme limits of the auricular cartilage. With the spread of the lesion, the tumefaction becomes less hard, imparting a boggy feeling to the palpating finger,—never that characteristic of fluctuation. Naturally the lobe is never involved, and this supplies a differential point between auricular perichondritis and a severe and extensive auricular inflammation of more superficial origin, in which the lobe is apt also to be involved. Fever and accompanying pulse changes are frequently present in some degree at the onset.

Terminations.—(A) The lesion may undergo resolution with absorption of pus or serum and a return to the normal. (B) There may be local recovery with considerable permanent thickening and deformity. (C) The presence and pressure of the pus may give rise to necrosis of the cartilage, with very great permanent deformity.

Treatment.—The treatment varies with the severity and extent of the lesion, and calls for the surgeon's best judgment. When the disease is apparently localized in a small area and shows no tendency to spread, the application of a wet dressing,—*i.e.*, of a saturated boric acid solution as hot as the patient can bear, and renewed frequently,—or of a 10 per cent. solution of ichthyol, may help to control the inflammation. When, however, a considerable effusion of fluid has taken place beneath the perichondrium, it is safer to sterilize the part as for a major operation and, with the patient under a general anæsthetic, make a free incision through the perichondrium. After the fluid has been evacuated, a very small gauze drain should be introduced into the wound and for a short distance

between the cartilage and perichondrium, and over this pressure applied by means of a pad of sterile gauze, this being reinforced by a larger gauze dressing. This dressing should be changed daily, and particular care taken to prevent reaccumulation of serum or pus beneath the perichondrium. If this operation is unduly delayed, more radical measures may become necessary through necrosis of the cartilage.

So long as the inflammation is confined to the perichondrium—*i.e.*, so long as the cartilage is not directly involved—through-and-through drainage by an incision passing through the auricular cartilage is not called for. Very great swelling of the auricle is often seen as a result of inflammatory infiltration of the perichondrium plus an effusion of fluid between it

Fig. 80.—Auricular perichondritis.

Fig. 81.—Deformity resulting from neglect of lesion shown in Fig. 80.

and the cartilaginous plate. In such cases incision of the cartilage does not seem called for. If, however, after incision of the perichondrium, the exposed cartilage is found to have become involved and to have undergone necrosis, there should be no delay in dissecting from it its perichondrial covering and exsecting the necrotic area. Unless this is done promptly, the whole cartilaginous frame may become necrotic, with ultimate very unpleasant permanent deformity.

Hæmatoma Auris (Othæmatoma).—This describes a condition in which, as a result of local injury, there has occurred an effusion of blood between the auricular cartilage and its perichondrium. It occurs only on the anterior surface of the cartilage. At first usually localized, it may spread later so as to separate the perichondrium from the entire anterior surface of the auricular cartilage.

Etiology.—This is somewhat obscure. Apparently the aged and more especially the insane are particularly prone to certain degenerative changes in the auricular cartilage and perichondrium, in which condition hæmatoma auris is apparently easily induced by very slight injuries. Its occurrence

in this class of individuals has been noted with particular frequency in Germany, and the occurrence there of certain cases in which no history of trauma could be obtained has led to the belief that a certain number of cases occur spontaneously. One wonders, however, whether even in such cases—depending unquestionably in some degree upon degenerative tissue changes—there has not been some slight injury which, though overlooked at the time, has been the directly exciting cause. In this country, so far as the writer knows, the condition is almost always due to direct violence. In twelve years of almost daily attendance at aural clinics and hospital wards, no single case has come under his observation which could not be directly traced to local injury. The commonest cause is a fist blow on the ear, but it has occurred also as the result of a hard slap and of violent twisting or pulling of the ear. The accompanying illustration is of a prize-fighter in whom the lesion resulted from a blow (Fig. 82).

Fig. 82.—Hæmatoma auris, resulting from a blow upon the ear.

Symptoms.—Following the injury, a tumor upon the anterior surface of the auricle is rapidly formed. At first localized, it may spread rapidly upward and backward to the helix, and downward to the upper boundary of the lobe. The tumor is generally purplish or bluish red, its local temperature is usually elevated, and it presents the peculiar boggy feeling characteristic of fluid confined beneath the perichondrium. If very small and localized, the pain may not be severe. With an effusion of considerable size, the pain is usually intense. Insomnia due to pain, fever, and constitutional depression is apt to be present in severe cases.

Terminations.—(1) With a small effusion there may be complete and perfect local recovery. (2) The lesion may undergo resolution, part of the blood being absorbed and the remainder being organized into new connective tissue, which results in permanent thickening and deformity (Fig. 83). (3) Infection may occur, giving rise to destruction of the cartilage, and ending in final recovery with great permanent deformity.

Treatment.—When the collection of blood is small, shows no tendency to increase, and is not accompanied by excessive pain, it is well to depend largely upon time to bring about a cure. From the nature of the lesion, it is difficult to see how the application of lotions can be of value, and pressure or manipulation may obviously do harm.

In the case of a large effusion of blood, the indications for treatment are well suggested by the statement of Richard Lake, of London, that spontaneous recovery "is not certain to take place, but if it does, deformity is sure to result." In such a case a free incision—coextensive with the extent of the effusion—should be made through the perichondrium. A puncture is

worse than useless. The blood or coagulum should then be evacuated, a curette if necessary being used for the removal of firmly organized clot, and the space between perichondrium and cartilage should be lightly packed with sterile gauze. This dressing should be changed daily for several days, after which it may be omitted, and the parts pressed into apposition and held so by a gauze pad and dressing. If, as advised by some surgeons, the cavity is not packed, but is at once subjected to pressure, reaccumulation of blood is more than likely to occur.

Differential Points between Auricular Perichondritis and Hæmatoma Auris.—While the two lesions present clinically many similar features, they may usually be differentiated by consideration of the following points: (A) History: Auricular perichondritis is usually traceable to a localized point of infection,—*e.g.*, furuncle, lacerated wound, plastic surgery involving the perichondrium. Hæmatoma auris is usually traceable to direct violence,—*e.g.*, a blow. (B) Rapidity of development: Following a local infection, the development of auricular perichondritis is comparatively gradual and slow. Following an injury, the development of hæmatoma auris is exceedingly rapid. (C) Auricular perichondritis under transillumination shows no marked color differences between involved and uninvolved areas. In hæmatoma auris transillumination gives a dark purplish color over the area of the blood collection or clot.

FIG. 83.—Final permanent deformity resulting from hæmatoma auris.

Lupus. — Tubercular lesions of the skin covering the auricle, while probably fairly common in dermatological practice and clinics, do not come very often before the aurist. Only in cases giving rise to considerable swelling and deformity of the auricle (lupus hypertrophicus, lupus tumidus) is the aurist likely to be called in. The student will, therefore, find more authentic descriptions and directions for treatment of these lesions in works on dermatology.

The commonest form (lupus vulgaris), when attacking the auricle is usually seen simultaneously upon other parts of the face. The lesions appear as distinct papules or tubercles, reddish or brownish in color, which may later be covered with dry, brownish scales. A characteristic feature is the tendency to form permanent depressions, the substance of the tubercles apparently in some cases being absorbed or shrinking without erosion of the overlying skin. Between these depressions are often formed small cicatrix-like bands. The disease is distinguishable from a syphilitic skin lesion of somewhat similar appearance (a) by absence of

PLATE V.

FIG. 84.—Lupus hypertrophicus auriculæ.

a history or evidence of syphilitic infection, (b) by their slower development, and (c) by the greater tendency of the syphilitic papules to ulceration.

Treatment.—The treatment calls for constitutional building up of the patient by constructive tonics. The local treatment aims at the actual destruction of the diseased tissue forming the basis of each tubercular nodule. For this purpose, the nitrate of silver stick, the sharpened point of which is forced down into the core of each nodule, is said to be effective. Actual surgical removal of the tubercles, the denuded surface being later covered by skin-grafts, is also recommended. The skilful local use of the X-ray is said also to exert a curative action on tubercular skin lesions.

Lupus Tumidus (Lupus Hypertrophicus).—According to Politzer, this form of tubercular lesion may result by the following process from the more common variety (lupus vulgaris). The typical superficial tubercles, or nodules, becoming confluent, undergo surface necrosis, giving rise to a tubercular ulcer. Within the depths of this ulcerated surface are deposited newly formed tubercular nodules. Following this there is a moderate proliferation of firm granulations which bleed easily. This may result in great thickening or enlargement of the involved portion of the auricle, which usually includes the lobule. The accompanying illustration (Fig. 84) represents a lesion of this nature. Unless brought under fairly prompt control, the cartilage may become involved, and, should it undergo necrosis, great permanent deformity is likely to result.

In rare cases a tubercular lesion of the auricle assumes the form of a tumor of considerable size. Tubercular lesions of this form involving the auricle are apparently much commoner in certain parts of Europe than in this country.

Treatment.—The treatment recommended for this lesion is prompt and thorough removal of the diseased tissue. The tubercular granulations and nodules are subjected to vigorous curettage with a sharp spoon curette until healthy tissue is reached. This surface should then be painted with a strong solution of silver nitrate, 200 grains to the ounce, in order to destroy such tubercular tissue as the curette has failed to remove. The parts are then dried and covered with a protective gauze dressing. The above treatment may have to be repeated several times before the disease is eradicated. Finally skin-grafting may be practised in order to hasten the healing process and lessen cicatricial contraction. In severe lesions some deformity is probably inevitable, but is less than if the lesion is allowed to involve the cartilage before treatment is instituted.

Syphilis of the Ear.—Primary syphilitic lesions of the auricle, while a few cases have been recorded, are exceedingly rare. The writer in twelve years of attendance in aural clinics and hospitals has not met with a single case. Secondary lesions, however, in the form of papular or pustular eruptions, coincident with similar lesions upon the scalp, forehead, or other parts of the body, are said to be of fairly common occurrence in dermatological clinics.

The severe lesions—*i.e.*, deep ulcerations and condylomata—are more

often seen within the meatus. While the physical appearances may be most suggestive, their specific character can be positively determined only by the association of a reliable history of syphilitic infection, the presence of other evidences of constitutional syphilis, or possibly by a positive Wassermann reaction. The condylomata are described as "grayish-red, ragged, watery excrescences," firmer than the ordinary form of inflammatory granulation tissue, and tending to spread with great rapidity.

The formation of gummata upon the auricle or within the meatus is said to be exceedingly rare.

A final and confirmatory diagnostic test of these lesions is found in their response to antisyphilitic treatment.

The treatment is constitutional rather than local. The local treatment aims chiefly at cleanliness. The chief reliance, however, is to be placed upon the constitutional remedies. Mercurial injections or inunctions with potassium iodide in appropriate doses usually exert a fairly rapid influence upon the local lesion. Salvarsan is even more rapid and positive in its results. Probably the best results will be obtained by fairly large and repeated injections of salvarsan in combination with some form of mercurial medication. For a general consideration of the value of salvarsan in aural therapy the reader is referred to a later chapter dealing particularly with the influence of that drug upon labyrinthine and auditory nerve lesions.

Herpes Zoster Auriculæ; Herpetic Inflammation of the Geniculate Ganglion (Hunt).—The occasional appearance of herpes upon the auricle, with the coincidence of aural symptoms of varying severity, has long been known to occur, but only since the publication of Dr. J. Ramsay Hunt's papers on the subject have we possessed any definite conception of the nature of the lesion. We now recognize the symptoms as directly due to an inflammation of the geniculate ganglion. According to Hunt, the zoster zone of the geniculate ganglion is sharply confined to the membrana tympani, the walls of the auditory meatus, the concha, tragus, antitragus, antihelix, and helix, rarely extending beyond the boundaries of this area. The herpetic eruption (Fig. 85) may occupy the whole or only a small portion of the geniculate zone. The appearance of the herpes is occasionally preceded by rather severe pain referred to the ear and mastoid region. With the development of the herpes there may be considerable swelling of the canal and pinna.

The symptoms may be limited to the aural pain; or the inflammation may spread from the ganglion to auditory and facial nerves, in which case there may be marked disturbance of hearing (cochlear branches of 8th nerve) or symptoms of vestibular irritation,—*i.e.*, nystagmus, vertigo, vomiting, etc. The latter phenomena point of course to involvement of the vestibular branches of the 8th nerve. Since the connection of the geniculate ganglion with the 7th nerve is far more direct and intimate then with the 8th nerve, facial paralysis is far more frequent than are the symptoms due to disturbances of the 8th nerve, with which the geniculate

PLATE VI.

FIG. 85.—Herpetic eruption due to inflammation of the geniculate ganglion (Hunt).

ganglion communicates chiefly through the small connecting branch known as the *pars intermedia*.

It is of the greatest practical importance that aurists should be prepared to recognize this lesion, for the reason that in a severe case the physical signs may strongly resemble those of a severe tympano-mastoid inflammation, in which rupture of the drum-head has not occurred.

The history of aural pain, quickly followed by herpes confined to the geniculate zone and the frequent occurrence of paresis or paralysis of the muscles supplied by the facial nerve, should enable the careful observer to arrive at a correct diagnosis. A careful reading of the papers referred to below is advised.[1,2]

Malignant Disease of the Auricle: Epithelioma; Cancer of the Auricle.—Epithelioma of the auricle is not very common and, when it does occur, shows no distinctive features directly attributable to its situation. That is to say, it presents the characteristic features common to such growths in whatever region they may appear. Epithelioma originating at some point upon the auricle grows rapidly, and the overlying skin is quickly eroded. The auricular cartilage is soon involved and may be very rapidly destroyed.

As to the surgeon's responsibility in the management of these cases, there is one point which can not be too strongly emphasized,—viz., that an epithelioma originating upon the auricle may in the first stage be removed surgically with good chances of a permanent cure. If surgical relief is long withheld, however, the neoplasm may suddenly exhibit very rapid growth, spreading quickly through the auditory canal to the tympanum, to the parts in front of and behind the ear, and finally may attack and destroy a large portion of the temporal bone. When such extensive involvement has taken place, recurrence is likely to follow any operation which may be employed.

INFLAMMATORY DISEASES OF THE EXTERNAL AUDITORY MEATUS.

Acute Circumscribed External Otitis; *Furunculosis of the Meatus.*—A furuncle in the meatus is one of the most painful, as it may be one of the most troublesome, conditions which the aurist is called upon to treat. It involves only the outer, or fibrocartilaginous, portion of the canal. It may be single or multiple. It occurs most frequently upon the posterior wall or floor, next upon the roof, and least frequently on the anterior wall.

Etiology.—*Predisposing Causes.*—The number of cases in which the same person in different years has suffered from recurrent furuncles of the meatus, and the still greater tendency of the lesion to immediate recurrence, impel one to believe that there are certain predisposing factors—not always easy to determine—which render certain individuals pecu-

[1] Hunt, J. R.: Herpetic Inflammation of the Geniculate Ganglion; Jour. of Nervous and Mental Diseases, February, 1907.

[2] Hunt, J. R.: Further Contribution to Inflammations of the Geniculate Ganglion; Amer. Jour. of the Medical Sciences, August, 1908.

liarly susceptible to the disease. People, for example, having the *gouty or rheumatic diathesis* seem to be particularly frequent sufferers. *Depressed constitutional states—e.g.*, lowered vitality caused by insufficient food or unhealthful surroundings or modes of life—are undoubtedly factors, as is shown by the relative frequency of furuncles of the meatus among the poorer class of dispensary patients as compared with those seen in private practice. It occurs, however, in all classes. *Chronic eczematous affections* of the concha or meatus also predispose to the development of furuncles, probably by affording denuded points for the entrance of germs.

The *exciting cause* is always a local infection. This may be brought about by a chronic purulent discharge from the middle ear acting upon a denuded area in the meatus. Quite often the lesion may be traced to the habit of scratching the canal wall with a toothpick, hairpin, or even with the finger-nail, this resulting in an abrasion which provides the point of entrance for infective matter.

Symptoms.—The symptoms of which the patient complains may be mentioned in the following order: (a) Sensitiveness to slight manipulation; (b) pain; (c) tinnitus aurium; (d) impairment of hearing. Discharge, when present, occurs so late in the disease, and is so frequently absent, that it can hardly be regarded as a characteristic symptom.

The patient may first be conscious of a sense of occlusion, or "stuffiness," in the affected ear, and with this he may notice that very slight manipulation—*e.g.*, as in drying the ear after the morning bath—gives rise to a sense of soreness or even a sharp pain in the ear. Later this discomfort or pain may be induced by extensive movements of the jaw, as in yawning. The next symptom is usually that of constant pain in the ear, at first of moderate grade, but gradually increasing and finally becoming unbearably severe. The pain caused by a furuncle of the meatus is in many cases quite as severe as that accompanying acute otitis media, and is augmented by the intolerable sense of soreness usually present. A characteristic feature of this pain is found in the fact that it is usually made worse by movements of the jaw,—*e.g.*, chewing, yawning, etc. This is, of course, explained by the close anatomical relation of the cartilage of the temporomaxillary joint and the cartilaginous framework of the meatus. This phenomenon, when present, is pathognomonic either of acute circumscribed or acute diffuse external otitis, and clearly differentiates these lesions from acute inflammatory conditions confined to the middle ear. Tinnitus aurium is a symptom the prominence of which varies in different cases from slight head noises, that are overlooked in the severity of other symptoms, to loud ringing noises which add much to the patient's discomfort. In some degree it is nearly always present. Impairment of hearing is not usually present at the onset, but becomes progressively more noticeable as the calibre of the canal is reduced, and may be very marked if the occlusion of the canal is complete.

Physical Examination.—Palpation comes first as a means of determining the presence of a furuncle in the meatus, for the reason that it will

in many cases elicit tenderness before changes in the contour and color of the canal wall can be detected. The finger should be pressed firmly against the cheek above and in front of the auricle, and brought downward along the line of anterior attachment so as to press the tragus inward. This will surely elicit tenderness if there is a furuncle in the anterior canal wall, and often even though it may be situated elsewhere in the fibrocartilaginous meatus. If, however, no pain is caused by pressure on the tragus, the cartilaginous meatus should be moved in different directions,—*i.e.*, forward, upward, and downward. If this procedure causes no pain, we may be quite confident that the disease is not located in the fibrocartilaginous canal.

Inspection by Reflected Light.—In the earliest stage of a furuncle in the canal, inspection may not reveal any localized swelling nor any changes in the color of the overlying skin. In this case the different walls of the canal should be subjected to pressure by means of a cotton applicator wound with cotton. In this way we are usually able to determine the point of local tenderness, which coincides, of course, with the point of infection. Later, inspection will reveal one or more points of localized swelling, according to whether we have to deal with a single furuncle or with a multiple lesion, furunculosis. With the progress of the lesion the canal is always, therefore, narrowed in one or more of its diameters, in accordance with the involvement of the different walls. In severe cases the swelling may be so great as completely to close the canal. Even in such cases, however, it may be possible to introduce a very small speculum beyond the furuncle and thus bring the drum membrane into view. Whenever possible without the infliction of too much suffering, this should be done. Careful inspection of the drum membrane is important for the reason that, as soon as we can determine that the membrana tympani is not acutely inflamed, we are in a position to exclude positively the middle ear as the site of intercurrent suppurative inflammation, and this without inspecting the drum membrane we are not able to do.

Course of the Disease.—In some cases a single furuncle will rupture spontaneously into the canal, and this may, or may not, determine a favorable course toward recovery. Oftener than not, such spontaneous rupture does not provide adequate drainage, and the inflammatory process continues to spread, foci of infection (*i.e.*, furuncles) developing simultaneously upon other walls of the canal. Quite frequently spontaneous rupture is so long delayed as to cause the patient excruciating pain, the inflammation finally extending to surrounding structures.

Extensions of Inflammation.—The involvement of structures about the ear depends somewhat upon the site of the furuncle. (A) With a furuncle upon the *anterior* wall, there often occurs very marked swelling of the tissues in front of and below the tragus. In such cases the parotid gland may be distinctly involved. (B) With a furuncle confined to the *posterior* or *posterosuperior* canal wall, there is not infrequently very considerable œdema over the mastoid region immediately behind the ear.

This commonly results in more or less complete obliteration of the post-auricular sulcus and the production of a very characteristic deformity,—the auricle being displaced forward, or forward and downward, so that it stands out from the head in striking contrast with the opposite sound ear (Fig. 86).

Extension of inflammation may also take place through the deeper structures of the auricle itself,—*i.e.*, it may cease to be in any sense a circumscribed inflammation, and spread widely between the perichondrium and cartilage, giving rise to severe and extensive suppurative perichondritis (Fig. 87).

Fig. 86.—Displacement of left auricle resulting from a furuncle in the posterosuperior canal wall.

Fig. 87.—Necrosis of auricular cartilage and deformity resulting from extension of infection from a furuncle in the canal. (Dr. Held's case.)

With a severe infection of the fibrocartilaginous canal, it is not unusual to find the lymphatic glands of the neck, particularly those in front of and behind the sternomastoid muscle of the affected side, enlarged and somewhat sensitive to pressure.

Since swelling behind the ear with consequent displacement of the auricle may occur with acute suppurative mastoiditis as well as with furunculosis of the meatus, it may be well to mention here three points which should aid us in differentiating between the two conditions causing this deformity:

Postauricular Œdema due to Furuncle of the Meatus.	*Postauricular Swelling due to Acute Suppurative Mastoiditis.*
1. Drum membrane usually intact, and may be normal or only slightly congested.	1. Drum membrane usually perforated, and always shows some of the cardinal signs of suppurative otitis media.
2. Pressure upon tragus and manipulation of auricle causing movement of fibrocartilaginous meatus usually cause severe pain.	2. Pressure upon tragus and manipulation or movement of fibrocartilaginous canal cause absolutely no pain.

3. Firm pressure over the mastoid process just behind the postauricular sulcus and opposite the orifice of the meatus will, if so directed as not to disturb the position of the auricle, cause absolutely no pain. The œdematous tissues pit deeply under the compressing finger, after which no bone tenderness can be elicited. Pressure at exactly the same point, but directed slightly forward so as to disturb the auricle, causes severe pain.	3. Firm pressure upon the mastoid process just behind the postauricular sulcus and opposite the orifice of the meatus usually elicits deep-seated bone tenderness.

The practical diagnostic value of the above is well illustrated by the following case. The author was asked by a general surgeon to confirm a diagnosis of mastoiditis in a hospital patient upon whom he was about to operate. The patient, a man of thirty years, presented superficially the following conditions, upon which the diagnosis had been based: (1) he was suffering severe earache; (2) there was very marked œdema behind the left ear and the postauricular sulcus was obliterated; (3) the auricle was displaced forward and stood out from the side of the head in conspicuous contrast with its fellow of the opposite side. The parts about the ear were shaved in preparation for immediate operation. That the mastoid was not involved was clearly shown by the following facts: (a) the drum membrane, rather difficult to inspect, was found to be practically normal; (b) the posterior wall of the fibrocartilaginous meatus was intensely inflamed; (c) any manipulation of the auricle causing movement of the cartilaginous meatus caused intense pain; (d) firm pressure upon the mastoid, so directed as not to disturb the auricle, caused the patient no pain. I learned later that he made a perfect recovery under treatment appropriate to furunculosis of the meatus.

Prognosis.—The prognosis in this lesion is somewhat clouded by the pronounced tendency to recurrence. A single furuncle may appear to be progressing favorably toward recovery, only to be succeeded by a second furuncle on the opposite wall of the canal. Every aurist can recall cases in which the patient has been afflicted with a series of furuncles,—three, four, five, or more,—each following the other in the same canal and in rather quick succession. It is well, therefore, to acquaint the patient with this danger, and impress upon him the importance of following the prescribed treatment to the letter.

Treatment.—The marked tendency to recurrence would seem to prove a constitutional basis in many of these cases. Internal, or constitutional, remedies should, therefore, play some part in their management. The treatment in every case should begin with thorough cleansing of the alimentary tract. Further than this, it is well to divide these cases into two general classes,—*i.e.*, (1) those presenting evidences of lowered vitality or resistance, for whom simple or constructive tonics should be prescribed; and (2) the overfed, full-blooded, or plethoric class,—the type of individual to whom in the older text-books the term "apoplectic" was applied. Such an individual requires careful regulation of diet, restriction of alco-

holic drinks, and regulation of the bowels. For the latter purpose, the administration of small doses of rhubarb and soda three times a day before meals has proved very satisfactory. This rather empirical method of classifying and prescribing for patients suffering from furunculosis of the meatus, has seemed favorably to influence the average results.

That the local treatment of this lesion has proved a troublesome problem in otology is shown by the long list of unrelated therapeutic measures which have been proposed for its relief. Many of these remedies have travelled a long journey, passing through one text-book to another, but have found little place in actual practice. Among them we find the following: Abstraction of blood by the application of leeches in front of the tragus; abstraction of blood by the so-called artificial leech; application of heat (a) by means of poultices and (b) by fomentations,—*i.e.*, application of heated lotions; application of cold by means of the Leiter coil; introduction into the meatus of various antiphlogistic agents, among which is mentioned pig's fat impregnated with opium (Politzer), etc., etc. The writer believes that most of these remedies are worse than useless, and that we but obscure our view of the therapeutic field when we allow our attention to be diverted from the few tried therapeutic measures upon which practical experience has stamped her approval.

As the local treatment must necessarily vary with the condition present, it may be well to re-state briefly the four stages through which a furuncle may pass,—viz.:

(1) *Initial stage*, in which the patient experiences a constant sense of discomfort, but pain only on manipulation of the cartilaginous meatus. Inspection may reveal no noticeable change in the contour or color of the canal, but palpation with a cotton-wound applicator discloses a point of maximum tenderness, representing the focus of infection.

(2) *Stage of inflammatory infiltration*, in which inspection reveals circumscribed redness and swelling of one or more walls of the meatus. Palpation shows the tumefaction to be hard, tense, non-fluctuating, and exquisitely sensitive to pressure. This is the most painful stage of the disease.

(3) *Abscess Stage.*—The involved area, representing a circumscribed and encapsulated collection of pus, bulges into the canal, is found by palpation to be distinctly fluctuating, and may point and rupture spontaneously into the canal.

(4) *Stage of Surrounding Cellulitis.*—Certain cases fail to develop an encapsulated abscess, and extend by invasion of surrounding structures, giving rise to œdema or cellulitis in front of or behind the ear, with consequent displacement of the auricle.

Local Treatment.—In the *initial stage* our purpose should be to abort the attack. The entire canal should be thoroughly cleansed of cerumen, exfoliated epithelium, or other impurities. This may be done by first irrigating the canal with warm boric acid solution and then scrubbing it out by means of cotton-wound applicators dipped in 95 per cent. alcohol. After this a pledget of cotton saturated with a solution of carbolic acid,

1 to 100, should be placed in the meatus and allowed to remain for a minute, or until the part begins to tingle. This should leave the canal surgically clean. As a permanent dressing my preference is for a wick of sterile gauze which has been saturated with a carbolic acid solution, 1 in 300, and then wrung out nearly dry. This should be packed rather firmly into the outer (membrano-cartilaginous) part of the canal, the pressure thus maintained seeming to influence these cases favorably. The concha should be filled with a ball of sterile absorbent cotton, which may be held in place by collodion or adhesive plasters. This dressing should be changed daily, the canal each time being thoroughly cleansed and dried. This method of treatment seems in a fair proportion of cases to have the effect of dispersing a beginning infection and bringing about a cure. When, however, this happy result does not follow, the lesion passes into the second stage, and may demand more vigorous measures for its relief.

Second Stage.—In this stage the lesion is theoretically a surgical condition for which a free incision would seem the logical procedure, and this indeed may be ultimately necessary in any case reaching the stage of inflammatory infiltration. It is definitely established by experience, however, that a certain considerable proportion of cases are controlled, and recover perfectly, under the following simple plan of treatment. The canal is cleansed as described in the last section, or simply by irrigation with a warm boric acid solution. A fold wick of sterile gauze, moistened in some non-irritating solution, is introduced into the canal, not tightly, and cut off so as not to protrude beyond the orifice of the canal into the concha. This wick is to be kept moist (not soaked) by some bland antiseptic solution. For this purpose the proprietary preparation, Cresatin, gives excellent results. If this cannot be obtained, the following prescription has proved satisfactory in many cases:

℞	Acid carbolic................	gr. i
	Sodii bicarbonat.............	gr. xx
	Glycerin.....................	ʒi
	Aquæ, q. ad.	℥i

FIG. 88.—Furuncle knife.

The wick should be kept moist by adding only three or four drops by means of a medicine dropper every two hours or so, a ball or pledget of sterile cotton being placed lightly in the concha in the intervals. Under this treatment the inflammation and infection is in many cases seen rapidly or at least progressively to subside. If too much of the solution is used, so as to keep the wick soaked, maceration of the cutaneous covering of the canal is likely to occur, causing desquamation of the cuticle, with the added lesion of a diffuse and troublesome dermatitis. As soon as

the localized infection is controlled by the above means, it is better to discontinue the solution, merely keeping the canal lightly packed with a dry wick of sterile gauze for a few days.

If the treatment above outlined fails to control the infection, and particularly if the inflammation is seen to be progressive, a free incision becomes the most rational step toward relief. As this operation, though short, is exceedingly painful, it is always best to have the patient under nitrous oxide anæsthesia. The canal should be previously sterilized by peroxide of hydrogen, followed by irrigation with a warm solution of carbolic acid, 1 to 200. The point, or focus, of infection having been determined by inspection and palpation, the knife should be introduced into the canal beyond the inflamed area, and the furuncle freely and rather deeply incised as the blade is withdrawn. For this purpose a straight knife (Fig. 88) has always seemed to me more easily controlled, and therefore preferable to the curved bistoury frequently employed. While from the nature of the lesion the depth of the incision cannot be exactly gauged, our purpose should be (1) to have it so placed as to reach the centre of infection, and (2) to carry it through the inflamed cellular tissue and into the perichondrium without cutting deeply into the cartilage. It is, however, better to cut a little too deeply rather than to err in the other direction with an incision too superficial to reach the source of trouble. An additional and important advantage of having the patient under the influence of an anæsthetic is that it permits one to use pressure for the removal of pus, should it be present, or at least to make sure by the use of a probe that the incision is sufficiently deep.

After the patient recovers consciousness, which with nitrous oxide anæsthesia occurs within a few moments, it is well to irrigate the canal again with a carbolic acid solution, 1 in 200, as hot as the patient can bear it without too great discomfort. This second irrigation is distinctly useful, as the heat and the locally anæsthetizing effect of the carbolic acid reduce the sensitiveness of the part and facilitate the subsequent treatment. The canal should now be dried as thoroughly as possible and subjected to rather firm pressure by the introduction of a wick of sterile gauze which has been saturated with cresatin or a weak carbolic acid solution, 1 in 300, and then wrung out nearly dry. If such a patient can be confined to bed for a day or two, the efficacy of this treatment is likely to be enhanced, and in this case it is well to protect the ear with a large dressing of sterile gauze held in place by a bandage. The whole dressing should, of course, be changed daily.

With regard to the irrigation fluid to be employed in cases of furunculosis, the writer believes that bichloride of mercury, even in weak solutions, should never be used, since it is likely to produce a dermatitis distinctly favorable to the development of secondary furuncles. If for any reason the use of carbolic acid is not advisable, a boric acid solution is non-irritating and usually quite satisfactory. It is hardly necessary to state that carbolic acid in any solution stronger than that indicated in the prescription

on the foregoing page should never be advocated for home use by the patient or his family.

The practice of incising a furuncle, and then directing the patient to syringe the ear at stated intervals, is quite unsurgical, the residual water left in the meatus being apt to macerate the skin covering the healthy portions of the canal, a condition distinctly favorable to the growth of bacteria. Cases so treated will be found to result in a large percentage of recurrences, whereas in my experience immediate recurrence is not frequent when the dry treatment, as above described, is carefully carried out. Routine irrigation of the ears is admissible only in one class of patients suffering from furunculosis of the meatus,—viz., dispensary patients who can be seen only at such long intervals that no other method is available. In such cases the best results will probably be obtained by prolonged irrigation, frequently repeated, with sterile water as hot as the patient can bear it. In advising hot irrigations, the patient should be informed that he will be likely with each repetition to experience distressing vertigo, but that this discomfort will be short-lived and without serious consequences.

Third Stage.—When a distinctly fluctuating abscess has formed at the site of the initial infection, the lesion is often no longer excessively painful, and incision usually causes much less pain than in the second stage. Nevertheless, it is better, when practicable, to anæsthetize the patient, so that following the incision the pus may be thoroughly expressed and the interior of the cavity scraped out, if need be, by means of a small curette, or swabbed out with a carbolic acid solution, 1 in 20. A small drain may then be introduced into the abscess cavity, and pressure applied by means of a wick of sterile gauze.

In the *fourth stage* the treatment does not differ materially from that above described. It is very rarely necessary to incise the œdematous parts behind or in front of the ear, secondary abscesses in these regions being exceedingly rare. Incision at the point of infection in the canal usually relieves tension and provides an adequate pathway for the escape of pus. The application of a wet dressing over the adhacent œdematous areas may, however, prove of value.

In the foregoing pages the author has outlined the plan of local treatment which in his own experience has seemed to insure the best average results. The best plan of local treatment, however, leaves the patient who has suffered from this painful and depleting lesion particularly prone to recurrences.

Against these recurrences I believe that the patient's most effective defense will be found in the use of an appropriate vaccine. Naturally, whenever pus from the lesion can be obtained, an autoginous vaccine should be prepared. Under present methods, however, the great majority of cases of furunculosis of the canal are controlled without incision.

Stock Vaccines.—Though difficult to explain, it is a fact abundantly established by experience that the vast majority of cases of furuncles of the auditory canal are due to a staphylococcus infection. Utilizing this fact and the large clinical material of her clinics and wards, the Manhattan Eye and Ear Hospital of New York has for several years used for this lesion a stock vaccine made in her own laboratory. This is a composite vaccine, *i. e.*, made from various staphylococcus strains derived from pus taken from various patients suffering from this particular lesion. There is no question that this particular stock vaccine has great value in establishing an immunity against recurrence of this distressing lesion. For the rationale of this treatment the reader is referred to the short chapter on serum therapy.

Note.—Of recent years the writer has come to regard furunculosis of the meatus as a hospital condition. A few days of absolute rest in bed and the better general and local care which hospital control makes possible undoubtedly help to a quick and permanent recovery.

Acute Diffuse External Otitis.—This, according to my experience, is a comparatively rare condition. It exists whenever, in the absence of determinable foci of infection, the four walls of the canal are in a state of acute inflammation. It differs, therefore, from the occasional diffuse inflammation of furunculosis in the absence of the localized points of infection, single or multiple, which characterize the latter. Another difference between this and the circumscribed form of external otitis (furunculosis) is the frequency with which the whole canal is involved. That is to say, furunculosis of the meatus is almost invariably confined to the fibrocartilaginous portion, whereas in acute diffuse external otitis the canal in its entire length is usually more or less inflamed.

Causes.—Acute diffuse external otitis may occur as an accompaniment of chronic purulent otitis media, in which the discharge from the middle ear, being allowed to remain in the meatus, gives rise (1) to maceration and exfoliation of the cutaneous lining, and (2) acts as a direct irritant of the exposed subcutaneous surfaces. A more characteristic form of acute diffuse external otitis is seen as a result of direct violence, as not infrequently occurs in cases in which a physician with more zeal than skill has attempted with instruments to remove a foreign body from the meatus.

Symptoms.—The symptoms are very similar to those observed with furunculosis of the meatus,—*i.e.*, pain, sometimes very severe, increased by extensive movements of the jaw (yawning) and by any manipulations of the auricle which are communicated to the fibrocartilaginous meatus. Pressure upon the tragus always causes severe pain. Inspection by reflected light shows the calibre of the meatus to be greatly reduced by the diffuse swelling of its walls.

Treatment.—Since the treatment up to a certain point is the same as that outlined for furunculosis of the canal, repetition of details are not called for. Wet dressings by means of gauze and antiseptic solutions as advocated for acute diffuse external otitis, give satisfactory results in a large majority of cases.

Fig. 89.—Hyphomycetes, moulds: A, aspergillus nidulans; B, verticillium Graphii. (After Siebenmann.)

In a quite small percentage of cases, the inflammatory process will apparently yield to no treatment short of surgical intervention, which may be summed up in a few words:

(1) Thorough cleansing of the canal, after the method described in the treatment of furunculosis.

(2) One, sometimes two, longitudinal incisions, by means of the furuncle knife, through what may appear to be the more acutely inflamed wall or walls of the canal.

(3) Rather prolonged irrigation with a hot solution of boric acid, or carbolic acid, 1 in 200. The heat thus applied favors free bleeding, thereby relieving tension and tending further to cleanse the parts of bacteria.

(4) Careful drying of the canal, followed by the introduction of a wick of sterile gauze, or of gauze which has been impregnated with Dobell's solution, a solution of boric acid, or a sterile normal salt solution. The dressing should be changed daily, the canal each time being thoroughly cleansed. As soon as the inflammation is apparently controlled, better results are obtained by a dry dressing, the outer cartilaginous canal being firmly packed with a sterile gauze wick. Pressure by this means should be continued two or three days after the symptoms and inflammatory signs have disappeared.

Otomycosis.—This term is used to describe the condition in which some form of mould has found lodgement, and conditions favorable for its development, in the external auditory canal. It may be confined to the walls of the bony meatus, or may spread thickly over the drum membrane. The fungus may simply inhabit the meatus without causing any morbid changes in the tissues upon which it grows, in which case it gives rise to no symptoms whatever; and this is the condition found in over one-third of all cases (Bezold). In other cases the hyphomycetes may invade the tissues, the mycelia perforating the lining membrane of the bony canal and the skin covering the drum membrane, and giving rise to an acute inflammatory process.

Etiology.—Beyond the fact that the germ requires warmth and moisture for its growth, but little is definitely known as to the cause of its frequent occurrence in the ear. Oils and fatty matter form a suitable nidus for its development, and Bezold obtained in seventy-five per cent. of all cases examined by him a definite history of the patient's having instilled medicaments containing oil or glycerin into the ear.

Many different forms of mould have been demonstrated in specimens taken from the ear. Among them may be mentioned the *Aspergillus niger*, *Aspergillus flavus*, *Aspergillus nidulans*, and the *Verticillium graphii*.

Symptoms.—As before stated, the fungus may remain long in the meatus and produce absolutely no symptoms. As soon, however, as the mycelia invade the living tissues—*i.e.*, the skin covering the drum membrane or walls of the bony meatus—inflammatory changes are induced and the patient experiences more or less discomfort. The symptoms vary from an intense itching and sense of irritation to moderate—sometimes

rather severe—pain. The hearing may be impaired in cases in which the canal is more or less occluded.

Inspection by reflected light reveals a different picture according to the form of hyphomycetes present and the stage of their development. The drum membrane and the walls of the bony meatus may be covered with an adherent growth, the color of which may vary from very dark gray or black (*Aspergillus niger*) to brownish yellow (*A. flavus*); or it may be dark green in color (*Verticillium graphii*). Should inflammatory changes have taken place, the epithelium covering the drum membrane and canal walls may be thickened or exfoliated, or its removal may leave a red and inflamed surface.

Treatment.—Salicylic acid seems to retard or check the growth of the various moulds finding lodgement in the ear. The first step in the treatment is thorough mechanical cleansing of the parts. The mould should first be removed by means of cotton-wound applicators, and the canal walls and surface of the drum membrane then carefully scrubbed with 95 per cent. alcohol. As a further precaution the ear may be irrigated with a 1 per cent. solution of carbolic acid. Lastly the canal is filled with a 2 per cent. solution of salicylic acid in alcohol, which is allowed to remain in the ear five minutes or more. The patient is given a prescription for this solution (*i.e.*, 2 per cent. alcoholic solution of salicylic acid) with which he is directed to fill the canal night and morning, allowing it to remain from three to five minutes. This treatment usually controls the affection fairly promptly, but the patient should be seen occasionally during several weeks as a precautionary measure against recurrence.

When otomycosis occurs in an ear in which the drum membrane is perforated, the same line of treatment is usually effective. But since the tympanic recesses are not so easily reached, recovery is apt to be less rapid.

Croupous External Otitis (Otitis Externa Crouposa).—This is admittedly a rare condition. It may be defined as a diffuse inflammation of the inner, or tympanic, third of the meatus, in which this part of the canal and the surface of the drum membrane are covered by a fibrinous exudate. The membrane is repeatedly thrown off in the form of casts of the canal and drum-head. A new membrane soon forms. This cycle of exfoliation and re-formation may be repeated many times. The condition is somewhat anomalous in providing the only instance of a coagulated fibrinous membrane forming upon tissues not covered by mucous membrane.

Etiology.—Of this but little is definitely known. There are a few facts, however, which seem to me somewhat suggestive. It is known, for example, that the condition occasionally complicates furunculosis of the canal; that in the past it has occurred with comparative frequency in certain epidemics of influenza (a disease known frequently to cause blebs or bullæ of the canal); and that it may be engrafted upon the simple form of acute diffuse external otitis. It is possible that the development of the lesion requires some form of preëxisting acute inflammation plus the action

of some germ which has not yet been isolated. This theory may help to explain its gradual disappearance among the affections treated in aural clinics. That is to say, the disease during the past two decades has become exceedingly rare, a fact possibly due to the more general and efficient use of antiseptics within that period.

In a few instances the staphylococcus pyocyaneus and the streptococcus pyogenes have been found in the secretion or casts, but not with sufficient frequency to connect them as specific factors in the causation of the disease.

The SYMPTOMS are those common to acute inflammations of the canal,—viz., pain in the ear, often very severe,—increasing in severity during the formation of new membrane and subsiding as the membrane is dislodged. The hearing is naturally interfered with, and varies in accordance with the degree of occlusion of the canal. The course of the disease, though often protracted, usually ends in recovery.

The TREATMENT may be summed up in the word cleanliness. During the acute stage frequent irrigation with boric acid solution will help to hasten the formation and exfoliation of the membrane. During the periods between exfoliation and re-formation of membrane, dry treatment with insufflation of boric acid powder is said to give best results.

OBSTRUCTIVE CONDITIONS OF THE EXTERNAL AUDITORY MEATUS.

Foreign Bodies in the Meatus.—There is hardly a limit to the variety of foreign substances which may be introduced, either intentionally or by accident, into the external auditory canal. This is the experience of every aurist in hospital practice. Children furnish by far the greater number of patients. The writer personally has removed flies, bugs, glass beads, imitation diamonds, seeds and small fruit kernels, a bullet, a piece of chalk, and a piece of pencil lead.

In regard to the influence of a small foreign substance in the auditory canal, and the supposedly urgent need of its prompt removal, two facts should be impressed upon laymen and physicians alike,—viz., (1) that a small foreign body may remain in the canal for a long time, or even indefinitely, without injury to the ear and without even causing disturbance of function; and (2) that unskilful attempts at removal have frequently destroyed the usefulness of the organ, and have been the direct cause of not a few deaths.

REMOVAL OF A FOREIGN BODY BY MEANS OF THE SYRINGE.—Fortunately, the great majority of foreign bodies finding their way into the auditory canal can be removed without injury to the ear by means of the syringe. This statement applies to practically all smooth substances small enough to enter the bony meatus without having to overcome resistance at the isthmus, and to all smooth bodies remaining in the membrano-cartilaginous meatus which are not large enough completely to fill its lumen. The fountain syringe is not suitable for this purpose, a syringe with which the current force can be regulated by hand pressure being

infinitely more effective. Fig. 90 shows a metal syringe which can be sterilized by boiling and which answers this purpose admirably.

Before using the syringe the ear should be examined by reflected light to determine as nearly as possible the size, shape, and position of the substance to be removed. If small enough to be freely movable in the canal, a few injections of warm boric acid solution, the stream being directed a little upward and backward along the posterosuperior canal wall, will quickly expel it. When the foreign body has not passed beyond the isthmus and is so shaped and situated as to leave a demonstrable space between it and one wall only of the canal, the stream of water should be directed against this open space. In such a case, as soon as the water has filled the space between the foreign substance and the drum membrane, the principles of hydraulic pressure are brought into play, and the intruder is thrown out by the force of the increasing volume of water behind it. Were the stream not so directed, it might result in forcing the substance

Fig. 90.—Aural syringe.

into the depths of the bony meatus where its expulsion might be less simple. When a foreign body appears completely to fill the lumen of the membrano-cartilaginous canal, it is clear that irrigation will not dislodge it and may carry it further into the canal, unless the stream can be made to enter the space between the intruder and the drum membrane. When irrigation is employed in such a case, the auricle should be drawn outward and backward, and a rather forcible stream directed inward and a little upward and backward along the posterosuperior canal wall. Following this method, the water in most cases reaches the fundus of the canal, and the substance is brought away with the return current.

Insects.—When insects—*e.g.*, bugs—are seen crawling within the canal or upon the drum membrane, it is well before syringing to fill the meatus with olive oil. This dislodges them from their point of attachment and facilitates their expulsion.

Seeds, Peas, etc., within the Canal.—Much has been said of the danger in using the syringe of causing such substances to swell and tightly occlude the canal. The question to be considered in such cases is whether the pea or seed has already become swollen, or is so large as to render its

removal by irrigation doubtful; for in case of failure, the moisture left in the canal will be likely to render subsequent efforts at removal more difficult. When, however, the seed or pea is small and movable in the canal, there can be no possible danger in the use of the syringe.

While syringing does not act quite so effectively upon a substance in the depths of the bony meatus, there are in the author's opinion exceedingly few cases in which a foreign body lying free in any portion of the canal cannot by this method be removed without injury to the parts.

Cases Requiring the Use of Instruments.—While the syringe is effective in the vast majority of cases, conditions occasionally present themselves in which one may be obliged to resort to the use of instruments. In this category may be included the following:

1. Peas, seeds, etc., of such size, or so enlarged by the effect of heat and moisture, as to be no longer freely movable in the canal.

Fig. 91.

Fig. 92.

Fig. 93.

2. Solid substances which have become so firmly wedged in the canal (*e.g.*, at the isthmus or in the depths of the bony meatus) as to require mechanical dislodgement.

3. Solid bodies of irregular or angular shape, pressure of which is causing inflammatory swelling of the membrano-cartilaginous meatus. A deft use of instruments is often the only means of relieving this condition.

4. Solid substances which have been forced into the bony meatus, and are yet too large to repass the isthmus on the way out except in response to an equal amount of force. Children occasionally furnish such instances.

Choice and Use of Instruments.—In the author's opinion the safest instruments for removing solid substances from the ear are those typified in the accompanying illustrations.

The hook (Fig. 91) should present a terminal rectangular bend about 3 mm. in length, the extremity of which is rounded or dull. In using it the hook, or terminal bend, lying flat against the canal wall, is carried past the intruding substance. The handle, or shaft, is then rotated so that the hook squarely engages the foreign body, which can usually be easily withdrawn. This in my experience is a very practical little instrument which can be successfully employed in a majority of cases.

The dull ring curette (Fig. 92) can be used safely in only one way: it should be introduced either above or behind the body to be removed,—*i.e.*, between it and the roof or the posterior canal wall. The body is then crowded downward or forward against the floor or anterior wall, and thus pushed outward toward the orifice of the canal. Pressure with the ring curette from below upward is always dangerous, for the reason that it may force the substance backward upon, and even through, the drum membrane.

The author's adjustable ring curette (Fig. 93) is practically a dull ring curette in which, by sliding forward a movable rod, the ring can be made to assume a position at right angles to the shaft. Its application is obvious. The ring in the straight position is carried beyond the substance to be removed, and then changed into the rectangular position, in which it cannot fail to engage the intruder.

The writer has purposely not mentioned any form of forceps, because he believes that a forceps should never be used for removing a solid body from the auditory canal. The two main objections to its use may be stated as follows:

1. In removing a foreign body it is particularly important to avoid any injury to the canal. With care it is almost always possible to pass a small instrument between the intruding body and one wall of the meatus without injury to the latter. With the forceps, however, one has in effect simultaneously to pass two instruments—*i.e.*, the two forceps blades —between opposite sides of the foreign body and the corresponding canal walls. Unless the body is so small as to be obviously removable by the syringe, the danger of traumatism is doubled. Bleeding in case of failure renders further effort more difficult.

2. The chief objection to the forceps, however, is the danger of pushing the offending substance further into the canal. Not infrequently a solid, smooth substance, which seems easily and safely within the grasp of the forceps, slips from its blades and is driven further toward the drum membrane. Successive attempts may result not only in further displacement, but often in extensive laceration of the canal. The writer has known more than one case in which the drum membrane has been practically destroyed by this manœuvre, the foreign body being finally pushed into the tympanic cavity. Every experienced aurist has met with such cases.

Surgical Removal of a Foreign Body from the Meatus.—This is advisable whenever it becomes evident that the simpler methods above described cannot be employed successfully or without endangering the integrity of the organ. The conditions calling for operative intervention fall chiefly under two heads,—viz.:

(a) Inflammatory swelling of the canal so occluding its lumen that small instruments cannot be manipulated within the meatus without danger of injury to the deeper parts. Under such conditions the attempt to remove a foreign body through the meatus might result in irreparable injury to the ear.

(b) The aurist has occasionally to deal with cases in which ill-advised or unskilful attempts at removal have resulted in such traumatism of the canal walls that the exact size and position of the foreign body and the condition of the tympanic structures cannot be determined. Under such conditions further attempts at removal through the canal are apt to result in further injury.

It should be accepted as an axiom in practical otology that no instrument should be introduced into the meatus except under inspection by reflected light, and under conditions permitting one to know positively its exact position after it has entered the bony canal. Obviously, when the canal is swollen or bleeding, or its walls torn and lacerated, the use of instruments cannot be controlled by the recognition of familiar landmarks. Under such circumstances the attempt to carry an instrument beyond a foreign substance may result in serious injury to important tympanic structures, or even in dislocation of the stapes from the oval window. Bezold is authority for the statement that such accidents have been responsible for many deaths.

Students of otology will do well to bear in mind and adhere strictly to the following rule: *Whenever the membrano-cartilaginous canal is acutely inflamed, no attempt should be made to remove a foreign body even from the outer, or membranous, portion of the meatus, without having the patient under the influence of a general anæsthetic.* With the patient anæsthetized, it is usually possible, even in the presence of moderate inflammation, to remove through the canal a foreign substance which has not passed into the bony meatus.

The steps of the operation for removing a foreign body from the depths of the bony meatus are as follows: A curvilinear incision, following the curve of the auricular attachment, is made over the mastoid process. This incision passes through all the soft tissues, including the periosteum, down to the bone. The periosteum is carefully elevated anteriorly, and the lining membrane of the bony canal separated from its walls by means of a small curved elevator. Under traction the membranous canal is usually torn from its attachment to the annulus tympanicus and the drum membrane, and the auricle is folded forward upon the face, drawing with it the membranous canal out of the bony meatus. It is now an easy matter to remove the foreign body with forceps or other instrument, unless it has been forced through the drum membrane by previous manipulation, and lies hidden within one of the recesses of the tympanic cavity. After the intruder has been removed, the parts, having been re-sterilized, are replaced in position, and the posterior wound sutured. The membranous lining of the bony canal is held in place by a light packing of sterile gauze. If asepsis has been carefully observed, the wound resulting from this operation usually heals promptly.

In certain cases it will be found that, as a result of previous ill-advised attempts at its removal, the foreign body has been forced through the drum membrane, and therefore lies within the tympanic cavity proper.

Under such conditions it may be necessary to enlarge the opening in the drum membrane or even to remove it in order to locate the object which has been the cause of so much needless injury. In two cases operated upon by the writer, the drum membrane had been torn and lacerated beyond any hope of repair, before the patient came under his care.

Removal of Cerumen from the Meatus.—The secretion of cerumen, or "ear-wax," is confined to the area of distribution of the ceruminous glands in the outer part of the membrano-cartilaginous meatus. In amount it varies in different individuals from a very thin layer lining the outer end of the canal to large masses completely occluding its lumen. In a certain proportion of cases there is practical absence of cerumen so far as the eye can detect. These differences may occur in ears which are otherwise perfectly normal. Ordinarily the cerumen is confined to the outer, or membranous, portion of the canal. In some cases, however, in which there is no automatic elimination or extrusion of the cerumen formed, the last to be secreted pushes the previously formed mass further inward toward the drum membrane, and this process, repeating itself, finally completely fills the meatus.

When present in the meatus in masses large enough to disturb or endanger the auditory function, cerumen becomes practically a foreign body, which should be removed. The method of removing it calls for but brief notice here, since it is effected by practically the same measures as have been described for removing foreign substances finding their way into the ear. In the vast majority of cases, even large masses of cerumen may be removed easily and without injury to the canal by means of the syringe. As occasional exceptions to this rule may be mentioned the following conditions:

(a) The presence of large masses of tightly impacted cerumen completely closing the membrano-cartilaginous canal. In such a case the stream of water from the syringe may play upon the obstructing mass, exerting no influence upon its position. If we can insinuate a dull ring curette or other small instrument between the roof of the canal and the cerumen, we may by crushing the latter downward be able to create a passage by which the irrigation fluid may reach the fundus of the canal. By directing the stream along this pathway, the cerumen will in many cases be quickly expelled. Should this fail, it may be necessary to introduce a hook, or the angular ring curette, into the space above described and remove the cerumen by direct traction.

(b) A second condition for which the syringe is inadequate is found in cases in which the whole canal from the external orifice to the drum membrane is filled by a solidly packed mass, probably representing the accumulation of years. Such an accumulation is usually composed of cerumen intermixed with layers of exfoliated epithelium, finally becoming welded into an exceedingly hard and impervious mass. This condition is made known to us first by the inefficacy of the syringe, and second by the sense of solidity and resistance which it imparts to the probe or curette.

In this condition I believe that it is not always best to attempt to remove the entire mass at one sitting. If we can succeed in passing a dull ring curette for a short distance between the canal roof and the obstructing body, we may be able to force the ring down into it, and thus by traction withdraw the entire mass, or a portion of it. If only a part of the mass comes away, we may proceed in like manner to remove piecemeal so much of what remains as can be dislodged without injury to the canal. As soon, however, as we find ourselves in danger of injuring the canal walls, it is better to desist. We now direct the patient to fill the meatus night and morning with a solution of bicarbonate of soda (gr. xx ad ℥i), and to irrigate the ear once daily with a warm solution of boric acid (ʒj to the pint). If in two days this has not resulted in clearing the canal, we shall in most cases find the mass so much softened that we shall have little difficulty in removing it.

In some cases the use of the syringe leaves the drum membrane red and inflamed in appearance, a condition which usually subsides rapidly.

Exostoses of the Osseous Meatus; Bony Outgrowths from the Walls of the Bony Canal.—But little is known of the etiology of these growths. According to Bezold, they represent a condition rarely or never developed before puberty. They are multiple oftener than single, appearing oftenest on the anterior and posterior walls. When one growth only is present, the anterior wall, near the attachment of the drum membrane, is the most common site. As seen by reflected light, they appear as small, rounded protuberances, covered by normal skin, into the lumen of the canal. Palpation with a silver probe shows them to be hard and rather insensitive to pressure.

It is rare for these growths to occlude the canal completely, and the hearing is not usually noticeably impaired. Only in the case of acute middle-ear suppuration do exostoses of the canal cause anxiety, and then only if they are sufficiently large to interfere with drainage or the proper management of the tympanic lesion.

Treatment.—Usually none. Unless occluding the canal or unfavorably influencing the course of a middle-ear lesion, removal is not indicated. Should conditions arise rendering removal imperative the method of accomplishing this depends upon the character of the growth. If it is attached to the bony canal wall by a slender pedicle, it may be an easy matter to detach it by a single light tap upon a narrow chisel, the edge of which is held in firm contact with the canal wall at the point to which the pedicle is attached. When, however, the growth is attached by a broad base, no attempt should be made to remove it through the membrano-cartilaginous meatus. In such a case it is far safer to make a curvilinear incision behind the ear and carry the auricle forward upon the face, separating and lifting the membrano-cartilaginous meatus out of the bony canal. The bony growth may then be removed by means of a small chisel or gouge, or it may be practical and safe to introduce a small sharp bone curette and remove it from within outward.

CHAPTER V.

CAUSES OF ACUTE TYMPANIC DISEASE.

The conditions which predispose to, or excite, middle-ear inflammation may give rise to the mildest type of tympanic congestion or to the severest form of suppurative otitis media. That is to say, it is not possible, in the light of our present experience and knowledge, to speak dogmatically of one group of etiological factors as producing the milder ear lesions and of another as responsible for the severer types of tympanic disease. It will, therefore, enable us to avoid much useless repetition, and also conduce to a clearer and more practical view of the subject, to speak first of the causes of acute tympanic inflammation in general. It will then suffice to mention with each recognized type of otitis media the causes to which it is most often traceable.

Predisposing Causes.—Of predisposing constitutional conditions but little may be said beyond the fact that conditions of lowered vitality render the individual more susceptible to suppurative middle-ear lesions than are the physically and constitutionally robust. Only in this way can be explained the enormous prevalence of suppurative otitis media among the ill-nourished and poorly housed children of the poorer class of tenement-house dwellers in the large cities. The extent of this predisposition is not generally recognized either by the laity or by the medical profession, for the reason that, when drainage from the ear has once been established by a large perforation in the drum-head, the little patients suffer no pain and usually no constitutional symptoms sufficiently pronounced to attract attention. Should any physician in large dispensary practice among children care to investigate the truth of these statements, he may do so by making systematic aural examinations in any large series of cases. Such a series of examinations, if the results were carefully noted and the number of cases examined were sufficiently large, would not only be instructive to him, but might form the basis of a record of considerable value alike to otologists and to physicians generally.

Age.—Children undoubtedly suffer more frequently from acute middle-ear disease than adults. This is due in part (a) to the influence of pharyngeal adenoids, which are oftener present during childhood than in adult life; (b) to the greater susceptibility of children to the acute infectious diseases which so often give rise to otitis media; and (c) to certain anatomical differences in the Eustachian tubes in infancy and young childhood as compared with the adult type. These differences are described in the chapter dealing with the anatomy of the conducting apparatus.

Nasal Obstruction.—Undoubtedly the most potent predisposing cause of tympanic disease is to be found in conditions interfering with nasal respiration. Here again, with regard to acute otitis media, children

are the greater sufferers. The presence in the nasopharynx of large masses of adenoid tissue, with or without great enlargement of the faucial tonsils, constitutes a constant menace to the ears, which can be corrected only by removal of the growth. The important relation of adenoids to tympanic disease is made clear by the following facts, which may be verified by any physician who will take the time and trouble to do so:

(1) A majority of all cases of acute otitis media in children occur in those suffering from adenoids.

(2) Conversely, examination of any series of children suffering from adenoids will show in a majority of cases some pathological condition in one or both ears.

(3) Children with large adenoid growths rarely pass through a severe exanthematous attack without aural complication,—*i.e.*, without some grade of tympanic inflammation. And,

(4) Conversely, children without hypertrophy of the pharyngeal and faucial tonsils, or in whom adenoids have been successfully removed, usually pass through the infectious diseases of childhood without middle-ear inflammation.

Among other lesions interfering with nasal respiration are the following, which more commonly give trouble in adult life: Marked deviations to one or the other side of the nasal septum; septal ecchondroses or exostoses (commonly called spurs); hypertrophic rhinitis. These conditions predispose primarily to catarrh of the nasopharynx and membrano-cartilaginous portion of the Eustachian tube, and secondarily to middle-ear inflammation.

Exciting Causes.—Acute Rhinitis.—Acute middle-ear inflammation occurs very frequently as an accompaniment or immediate sequela of acute coryza, the common "*cold in the head.*" The author has made a habit of questioning his dispensary patients, as well as those in private practice, as to conditions or causes to which an existing acute otitis might be traced. That it followed "a severe cold" is a very frequent answer, but even more numerous are the cases in which absolutely no data upon which to base a theory as to its causation can be elicited from the patient. These cases can hardly be regarded as idiopathic, and are probably to be explained as having followed a mild coryza, or nasopharyngeal catarrh, which has escaped the patient's memory. The writer believes that *more cases of acute otitis media result from a simple nasopharyngitis, acting upon an individual in whom strongly predisposing conditions exist, than can be traced to all other exciting causes combined.*

Acute Infectious Diseases.—Just what percentage of cases of the acute exanthemata give rise to acute aural disease has not been determined. The author, as aural surgeon to the Willard Parker Hospital (New York city hospital for infectious diseases), has had some opportunity of observing the aural complications of certain infections—notably in scarlet fever and diphtheria. Taking these diseases for purposes of comparison, he would say that at least 10 per cent. of all case of scarlatina and 5 per cent. of all cases of diphtheria develop some form of acute

tympanic disease. Were the ears of all children suffering from these diseases examined daily, there is little doubt that a larger percentage of cases of acute tympanic disease would be diagnosticated than is represented by those usually attracting the physician's attention. Measles gives rise to acute suppurative otitis media, often of severe type, and in a percentage of cases probably falling little short of that associated with scarlet fever.

As to the comparative average severity of the aural lesions complicating the various exanthemata, it may be said that the purulent otitis media associated with measles is of severe average type, and that the percentage of cases developing suppurative mastoiditis is particularly large. The tympanic inflammation complicating scarlatina comes next to that associated with measles in the frequency with which the mastoid cells become infected. Another very serious and characteristic feature of scarlatinal otitis media is the astonishing rapidity with which the drum membrane may be actually destroyed. The writer has seen cases in which the posterior segment of a perfectly normal drum membrane has undergone almost complete disintegration within a few days of the onset of a scarlatinal otitis media. The destruction of the drum-head is in some cases so rapid and extensive as to render it impossible, after the systemic disease has run its course, for the tympanic defect to be repaired and the middle ear to regain its normal condition. This explains the very large percentage of clinic patients, suffering from chronic suppurative otitis media, who trace their aural lesions back to an attack of scarlet fever.

As compared with measles and scarlet fever, the aural complications of diphtheria are both infrequent and mild. There is greater danger of extensive destruction of the drum-head than of rapid and serious involvement of the mastoid cells. As with other infectious diseases, however, different epidemics vary greatly in the type of aural lesions produced. While in New York during the past three or four years the aural complications of diphtheria have been of comparatively mild type, the author recalls certain seasons in which the disease gave rise to many instances of severe tympanic and mastoid suppuration. Among the further results of such lesions he recalls one fatal case of brain abscess, several cases of purulent leptomeningitis, and a still greater number of cases of infective sigmoid sinus thrombosis.

Grippe, or influenza, is a frequent cause of more or less severe purulent otitis media, which in certain epidemics shows a marked tendency to rapid involvement of the mastoid cells.

Typhoid fever is a comparatively rare factor in the causation of acute tympanic disease. The aural lesions are in no way distinctive, or different from those occasionally accompanying other wasting diseases. The end of the third week and throughout the fourth week are said to cover the period during which aural lesions are most likely to develop.

Parotitis, or mumps, happily seldom causes tympanic suppuration. When the ears do become infected as a result of this disease, the labyrinth as well as the tympanum is not infrequently involved. This form of

panotitis may follow the usual course of a suppurative labyrinthitis; or the vestibular symptoms may rapidly subside, leaving in their wake extreme or even total deafness. It is one of the unhappy consequences of this lesion that the deafness, if pronounced, is apt to be permanent. Partial restoration of the cochlear function is said in certain cases to have been observed. Usually no improvement occurs.

External Causes.—Most of these are conditions which give rise to nasal or nasopharyngeal congestion. Thus, sudden or prolonged exposure to cold or getting the feet wet may cause grippe, pharyngitis, or only an acute rhinitis, any of which conditions may lead to acute tympanic disease. Inhalation of irritating vapors may act in much the same way. Thus, the use of fast automobiles by exposing the individual to sudden chilling of the surface or to the inhalation of heavily dust-laden air, has been responsible for some cases of acute otitis media.

The Nasal Douche.—The nasal douche, so frequently advised by some rhinologists and in some clinics, is doubtless absolutely necessary in certain cases of ozæna or of advanced atrophic rhinitis. It is also, however, a not infrequent cause of acute otitis media. Cases of aural disease originating in this way are known to every aurist. The author knows of at least one case in which the use of the nasal douche was directly followed by acute suppurative inflammation with mastoid involvement of both ears. Operation upon both mastoids revealed pus and extensive caries throughout both processes. In this case the patient said that, on using the douche for the first time, he choked slightly and "felt the fluid go to the ears." It is certainly safer, so far as the ears are concerned, to prescribe a nasal spray (atomizer) in place of the douche in all cases in which the nasal douche is not absolutely necessary.

Swimming or diving in cold water is another prolific cause of acute aural disease, which brings many recruits to the aurist and aural clinic with each return of the swimming season. It is probable that this cause acts in two ways—viz. (1) through water taken into the mouth, some of which is propelled through the Eustachian tubes to the middle ear; and (2) by the impact of the water against the membrana tympani. It is to be guarded against, therefore, (a) by stopping the ears by means of a wad of moistened absorbent cotton, and (b) by care to avoid choking or violent efforts to expel water taken into the mouth or nose while under water.

In a general way the above conditions may be said to constitute the chief etiological factors in the various types of acute middle-ear disease. Their importance in otology is obvious. The aurist who relieves the symptoms of an acute attack, but takes no thought of precautions to prevent recurrences or the development of the insidious chronic processes which may follow, hardly fulfils his whole duty toward the patient. And this obligation can be met only by careful consideration of the underlying causes in each case of tympanic disease. If we consider what might be accomplished in the way of preventing aural disease, the responsibility of the general practitioner is quite as great. In no branch of preventive medi-

cine are the possibilities greater. If it were more fully realized that the presence of adenoids, when sufficiently large to interfere in any degree with nasal respiration, almost invariably causes tympanic congestion; that their removal minimizes the danger of tympanic infection; that the infectious diseases of childhood give rise to suppurative otitis media more frequently than acute articular rheumatism gives rise to endocarditis, it is clear that the prevalence of aural disease among people of all ages might be very appreciably reduced.

Occupation and Habits as Causes of Aural Disease.—That certain occupations and habits may predispose to, or excite, aural disease, there is no room for doubt. Recognition of this fact and observation of certain forms of aural disorder thus induced have led to their being spoken of as "occupational diseases." This is an unscientific term which, however, has served a useful purpose in directing attention to certain vicious influences which should as far as possible be guarded against. Certain occupations and certain habits predispose to, or cause, certain forms of aural disease, but there is no evidence that the diseases in question differ in pathology or symptomatology from the corresponding disorders due to other causes.

Among the habits or occupations which occasionally cause aural disease or disorder may be mentioned the following: (1) The habitual or frequent use of certain drugs, — *e.g.*, tobacco, alcohol, the coal-tar products (phenacetine, etc.). (2) Occupations in which the individual habitually breathes heavily dust-laden air,—*e.g.*, railroad engineers, firemen, conductors, and others employed upon fast-moving trains; chauffeurs; street-sweepers, etc., etc. (3) Occupations subjecting the individual to the constant or frequently recurring influence of loud noises,—*e.g.*, blacksmiths, boiler-makers, naval and field officers and men subjected to the reports of heavy ordnance, workers in factories who are subjected to a constant roar of machinery. (4) Occupations requiring sudden or prolonged changes of air pressure, (a) upon the drum membrane, as in the case of caisson workers, or (b) within the tympanum,—*e.g.*, cornet-players and performers upon wind-instruments generally. (5) Workers in substances containing certain systemic poisons,—*e.g.*, lead, arsenic, phosphorus, aniline, etc.

In consideration of the many who, though subjected to one or other of the above influences, escape wholly any untoward result, one is forced to assume that individual idiosyncrasy or especial vulnerability to certain influences, or substantial powers of resistance to the same, must constitute in some cases a determining factor. It is also necessary to assume that in many cases the causal factor may act in two ways,—*i.e.*, either upon the sound-conducting mechanism or upon the labyrinth or auditory nerve.

As to the varying susceptibility of different individuals to certain influences, tobacco and alcohol furnish notable examples. Thus, one man may be able to smoke habitually large numbers of cigars daily and to drink wine, beer, or whiskey in considerable amount without any determinable injurious effect upon his ears, while another suffers regularly from any excessive indulgence in either.

Tobacco in susceptible subjects may affect the ears injuriously in two ways,—*i.e.*, (a) by inducing venous congestion or catarrh of the tubal mucosa, this giving rise to alterations of intratympanic pressure and tension; or (b) more rarely, may induce a slow and insidious form of auditory-nerve deafness. A colleague of the writer who has been an habitual though moderate smoker of cigars has been obliged to give up smoking on account of the aural symptoms induced. He states that whenever he is tempted to smoke, even in moderation, he regularly experiences tinnitus aurium and a sense of fulness and occlusion of the ears, which as regularly disappear after a few days of abstention from tobacco. In such a case the aural symptoms are clearly due to a mechanical interference with the functional activity of the Eustachian tubes.

As to the influence of alcohol upon the ears, while it is generally admitted that alcoholic excesses may be injurious to the auditory as to other cranial nerves, a search of the literature yields little that bears directly on the subject. The most authentic and apparently typical cases reported are those of two university students observed by the late Professor Bezold. These patients experienced sudden, very marked loss of hearing. Careful functional tests demonstrated a type of deafness characteristic of labyrinthine rather than of tympanic disease. Interrogation elicited the fact that both patients had for some months been in the habit of consuming enormous quantities of beer. All indulgence in alcoholic drinks was stopped, following which there was a gradual but complete restoration of hearing power.

Characteristic alcoholic deafness is probably in most cases dependent upon a neuritis of the auditory nerve, which, if the cause is not removed, leads to degenerative changes and permanent deafness.

Occupations subjecting their followers to more or less constant breathing of dust-laden air—*e.g.*, railway employés, chauffeurs, workers in dust-filled factories, etc.—are naturally productive of nasopharyngeal and tubal catarrh and the type of deafness characteristic of the tympanic changes secondary to such lesions.

Occupations in the pursuit of which the individual is subjected to the ear-strain or shock of constant or very loud noises, give rise to two types of deafness, both of which are due to labyrinthine injury or disturbance. Blacksmiths, machine-workers, workers in factories in which a constant whirr of machinery is heard, suffer sooner or later a diminution of hearing which is of the type characteristic of a labyrinthine or nerve lesion. In the case of continuous or frequently recurring noises, it is supposed that the more or less prolonged irritation of the auditory nerve filaments leads in time to degenerative nerve changes of which the loss of auditory acuteness is a logical manifestation. The auditory nerve changes in these cases are probably in some degree analogous to those induced by Siebenmann and Wittmaack in their experiments upon animals. It will be remembered that these investigators, in experiments separately carried out, subjected animals to the continuous sounding of certain musical tones, with the result that pathologic changes were induced in certain definite structures of

the perceptive mechanism, the site of these changes varying with the musical tone employed. Substituting for the prolonged hearing of a single musical tone the constant repetition of loud noises irritating to all parts of Corti's organ, it is not surprising that morbid changes leading to impaired hearing should result.

The cases of sudden, and often very profound, deafness following very loud explosive sounds—*e.g.*, the report of a cannon, the explosion of a mine, etc.—are obviously of quite different origin from those above described, these latter being due most probably to hemorrhage into the labyrinth or to sudden paralysis of the eighth nerve. That the symptoms, in some cases at least, are the result of changes primarily involving the auditory nerve filaments within the labyrinth is evidenced by the fact that the vestibular branch is also frequently disturbed.

Occupations requiring sudden or prolonged changes of air pressure upon the drum membrane or within the tympanum, most frequently give rise to morbid changes confined to the tympanum. This is usually the case in occupations calling for frequent increase of intratympanic pressure,—*e.g.*, cornet-players, glass-blowers, etc. The aural lesions to which caisson-workers are subject are also in a majority of cases confined to the tympanum, but occasionally involve the labyrinth.

In regard to the aural disorders of caisson workers, a word should be said as to the two very distinct lesions which this occupation may inflict upon its followers.

1. The first, and fortunately the commonest, is due to the great increase of atmospheric pressure to which the tympanic mucosa is subjected. Unless the tubes are absolutely patent the drum membranes are forced inward. In either case the mucous membrane lining the tympanic walls and drum membrane is subjected to enormous uniform increase of pressure, which when he leaves the caisson is as suddenly relieved. These sudden changes induce in some cases a most aggravated form of tympanic venous congestion, characterized in some cases by ecchymoses into the drum membrane, or hemorrhage into the tympanum. The symptoms are tympanic in character, alarming to the patient on account of their sudden advent, but usually yielding to time and rational treatment.

2. The second and more serious lesion of the caisson worker is a labyrinthine disorder the pathology of which is somewhat obscure. According to the theory of those who have made a study of it (Alt, Heller, H. v. Schrötter), the disease is the result of sudden alterations in the composition and density of the blood. As a result of the sudden diminution of atmospheric pressure when the individual leaves the caisson, arterial pressure is increased to the point of causing labyrinthine hemorrhage. Another theory is that the sudden decompression incident to leaving the caisson results in the formation within the blood of gas emboli, lodgement of which within the labyrinth is the direct cause of the lesion. The symptoms are those characteristic of labyrinthine hemorrhage—vertigo, ataxia, deafness. In severe cases the deafness is profound and may be permanent.

CHAPTER VI.

THE SUBJECTIVE SYMPTOMS OF AURAL DISEASE.

There are just four symptoms referable to the ear of which a patient suffering from any form of aural disease, acute or chronic, may complain,—viz.: (1) aural pain, or earache; (2) impaired hearing, or deafness; (3) aural discharge; and (4) tinnitus aurium, or the subjective sensation of noises in the ear. One or more of these symptoms will be present in any aural disorder. Vertigo and ataxia, which are such important symptoms of certain disorders of the labyrinth, are purposely omitted from this list, which is intended to include only the commoner phenomena which the patient himself would recognize as of aural origin.

Before entering upon a discussion of the more important middle-ear lesions, it may be well to consider briefly these subjective phenomena, which convey to the patient the knowledge that his ear is diseased.

Aural Pain.—Earache is present at some stage of practically every case of acute middle-ear inflammation. It may also be present (though comparatively rarely) in certain stages of chronic non-suppurative middle-ear disease. It is an invariable accompaniment of acute inflammatory disease of the membrano-cartilaginous meatus, and it may occur in very severe type as a reflex phenomenon from lesions widely separated from the ear. For purposes of differentiation, all of these types will be briefly discussed.

Pain in Acute Otitis Media.—The earache of acute catarrhal or purulent otitis media is described as resembling the pain of a severe toothache,—*i.e.*, throbbing or drawing in character, subject to exacerbations of intensity,—but of greater severity. It is probably one of the most unbearable types of physical pain. Its onset is usually sudden. Following a few premonitory twinges, the pain quickly attains a degree of intensity which makes sleep impossible. It is not influenced by manipulation or movements of the auricle or membrano-cartilaginous meatus. It is due either to increased tension through engorgement of the tympanic vessels, or to pressure and distention by fluid (serum or pus) collecting within the tympanic cavity. It is, therefore, often of short duration, subsiding quickly after spontaneous rupture of the drum membrane. This fact suggests the clearest indication for its relief: *Free incision of the drum membrane almost invariably relieves the pain of acute middle-ear inflammation.*

Pain in Chronic Catarrhal Otitis Media.—Another type of ear pain which may puzzle even the experienced aurist is a form of paroxysmal earache of moderate grade which is far commoner in chronic catarrhal otitis media than is commonly recognized. The patient gives a history of more or less frequently recurring attacks of otalgia, which, however, do not seem ever to be as severe as the pain accompanying acute tympanic

disease. The drum membranes may present changes characteristic of chronic catarrhal otitis media, but no sign of acute inflammation. In the mouth and throat are found no lesions of which the ear pain might be a reflex phenomenon. Under such conditions careful testing of the hearing will in many cases show the progress of a chronic middle-ear inflammation, with the periodic extensions of which the pain is probably associated.

Pain in Furunculosis of the Meatus.—The earache accompanying this lesion presents certain fairly constant characteristics. It is commonly gradual in its development, beginning as a sense of soreness about the canal, which later changes into a constant, throbbing pain of very severe type. It has frequently this pathognomonic characteristic,—*i.e.*, that it is increased or intensified by wide movements of the jaw,—*e.g.*, in yawning, or even chewing. This practically locates the lesion in the membrano-cartilaginous meatus, though the lesion may be either circumscribed or diffuse. Naturally the pain is intensified by any manipulation of the pinna which moves the cartilaginous meatus. As compared with the earache of acute purulent otitis media, the pain of furunculosis of the meatus is more gradual in its development and often less quickly relieved. While the indication is clearly a free incision through the focus of infection, this does not always furnish immediate relief of the pain.

Reflex Aural Pain.—Patients seeking relief from very severe earache, in whom physical examination shows the ears to be practically normal, are by no means uncommon in otological practice. In such cases we must carefully examine the mouth and throat for acute lesions which may be the underlying cause. By far the commonest cause of reflex aural pain is dental caries. Every aurist has seen such cases,—*i.e.*, the patient suffering acutely from aural pain, the drum membranes being practically normal, and one or more teeth of the corresponding side of the jaw being carious. The writer recalls the case of an intelligent, but nervous, woman who rather indignantly combated the suggestion of dental caries as the cause of very severe earache, which, however, was not relieved until a necrotic tooth was removed. The only relief in such cases is obtained through the dentist.

Other lesions causing reflex otalgia are severe acute tonsillitis, peritonsillar abscess, malignant disease of the base of the tongue.

Impairment of Hearing. — Some degree of functional impairment, transient or permanent, is probably present in every case of tympanic disease. Unfortunately, very slight grades of impairment are commonly not recognized by the patient. This is the more unfortunate since the onset of chronic non-suppurative otitis media is the stage in which most could be done to prevent progress of the lesion and the development of disabling deafness later in life. It is important, therefore, that very careful hearing tests should be made in all cases of slight aural disorder.

In acute middle-ear inflammation the hearing tests are not so important during the attack as after it has run its course, when a careful functional examination may demonstrate a residual disturbance of hearing which

may be easily corrected by appropriate measures promptly applied. Neglect at this stage may pave the way for chronic changes resulting in marked deafness later in life.

Aural Discharge.—Discharge from the ear, resulting from tympanic disease, always signifies partial or complete destruction of the membrana tympani,—*i.e.*, a perforation which may vary from a pin-head orifice to practical loss of the membrana tensa. It may be scanty or profuse; serous or purulent, and may be tinged with blood; odorless or exceedingly offensive. Since the character of the discharge necessarily calls for discussion in connection with the various lesions, but little need be said here beyond the mention of certain points in which the discharge in acute otitis media usually differs from that of chronic middle-ear suppuration.

Discharge in Acute Otitis Media.—Bacteriologically it varies in accordance with the character of the infection,—*i.e.*, it may contain any of the pus-forming bacteria, or in a purely serous inflammation may be mucoserous and sterile. Usually, however, an aural discharge remains sterile only a short period after incision or perforation of the drum-head, —*i.e.*, it soon becomes infected by germs from without.

Except when allowed to remain long in the canal, the discharge in acute otitis media is usually without odor. Following incision of the drum membrane the discharge is often at first very moderate in amount, but rapidly increases during the first twenty-four hours thereafter. Following this initial period, the discharge is usually profuse, and remains so during several days, then showing gradual diminution, and finally, in favorable cases, ceasing altogether at the end of a period varying from one to three or four weeks.

The distinguishing features of the discharge in acute otitis media are explained by the fact that they depend upon a lesion characterized by absence of bone necrosis, and which shows a marked tendency to self-limitation. Hence the absence of odor, and the gradual cessation.

Discharge in Chronic Suppurative Otitis.—The discharge in chronic middle-ear suppuration may be very profuse or so scanty that the patient may be quite ignorant of its presence. Usually it is subject to variations in this respect, being at times exceedingly profuse and at other times barely noticeable. Rarely does it disappear wholly, however, inspection by reflected light showing more or less destruction of the drum membrane and a granulating surface in the depths of the tympanic space, from which foul-smelling pus may be removed by means of a cotton-wound applicator. The odor also varies, being in some cases unbearably offensive and a source of unhappiness to the patient and in others of a sickening but less pronounced character noticeable only when removed from the depths of the canal.

These, then, are the characteristic features of the discharge in chronic middle-ear suppuration,—viz. (1) its constant odor and (2) its persistency. As in the case of acute otitis media, the character of the discharge is ex-

plained by the character of the lesion,—*i.e.*, its odor being due to the presence either of carious bone or of necrotic epithelial elements, and its persistency to the progressive character of the disease, which, though quiescent at times, tends rather to further extension and involvement of adjacent structures than to final resolution.

In acute otitis media the appearance of an offensive odor in the discharge usually means either gross neglect—the pus being allowed to remain for days in the canal—or a change in the character of the lesion—*i.e.*, chronicity due to bone caries.

In chronic suppurative otitis media, complete and permanent disappearance of odor from the discharge would suggest possible elimination of the necrotic bone, which occasionally is thrown off in the form of minute sequestra in the pus, thus paving the way for final resolution. Such spontaneous cures undoubtedly occur.

Tinnitus Aurium.—Of all the symptoms which the aurist is called upon to relieve, tinnitus aurium is the most elusive and difficult to control. It may be caused by any form of tympanic or labyrinthine disease, and may result from other conditions not dependent upon disease of the auditory apparatus. We may cure a tympanic lesion, but fail to relieve the tinnitus. This is a disappointing experience which every aurist has met with.

In some cases tinnitus is the only symptom of which the patient is conscious, and there may be little else that is abnormal either in the physical or the functional condition of the ear. So baffling may such cases prove that it may not be amiss to devote some space to a theoretic discussion of the various conditions which may give rise to this elusive symptom.

Obviously the most useful classification of the different forms of tinnitus aurium would be one based upon the underlying lesions or disorders. I believe that most cases will be found to fall under one or other of the following heads:

1. *Obstruction sounds,* or noises due to occlusion or impaired mobility of some portion of the sound-conducting apparatus.
2. *Blood sounds,* or noises produced by the blood current in vessels in or near the ear, and due either to disturbances of the local or general circulation, or to abnormalities in the size, shape, or position of the vessels.
3. *Labyrinthine sounds,* or noises due either to structural changes in the cochlea, or to alterations—either increase or diminution—of intralabyrinthine pressure.
4. *Neurotic sounds,* or noises due to abnormal irritability of the auditory nerve.
5. *Cerebral sounds,* or noises due to abnormal conditions acting upon the auditory centres in the cerebral cortex.

Obstruction Sounds.—Under this general term we may include the numerous cases in which tinnitus aurium can be traced to its cause in a demonstrable lesion of the sound-conducting apparatus. In acute tubal catarrh, two symptoms chiefly engage the patient's attention,—viz.,

impaired hearing and tinnitus,—and both disappear as the function of the tube is re-established. Again, in acute purulent otitis media, after the pain has been relieved by incision or rupture of the drum membrane, three symptoms continue to disturb the patient,—viz., impaired hearing, the discharge, and tinnitus aurium,—and all of them may disappear as resolution advances. In such cases the evidence is apparently conclusive that the subjective sounds are the direct result of a disturbed balance of the conducting apparatus. In studying these simpler forms of tinnitus, Panse advanced the theory that the function of the conducting apparatus is not only the transmission of sound waves to the ear, but also the conduction from the ear of sounds which otherwise would act too strongly upon the perceptive mechanism. As supporting this hypothesis, he quoted the statement of von Tröltsch[1] that "all noises actually arising in or near the ear must necessarily act more strongly on the auditory nerve if the natural outward sound conduction be in any way diminished."

Another way of explaining this form of tinnitus would be to state that there are always present in the neighborhood of the healthy ear sonorous vibrations due to certain physiological processes—*e.g.*, the blood current—which normally do not reach the inner ear; that such vibrations, when they do reach the perceptive mechanism, must do so by bone conduction; that bone conduction is always increased when the mobility of the ossicular chain is impaired; and, therefore, that sounds accompanying the simpler middle-ear lesions are in many cases the result of normal sonorous vibrations, which by reason of increased bone conduction are permitted to reach and impress the end organs of the auditory nerve. Have a vibrating tuning-fork held in contact with the median line of the skull, until its vibrations cease to be appreciated as sound. It is still vibrating, but now so feebly that the sound is no longer heard. Now close both ears by a finger pressed in each meatus, and the sound of the still vibrating fork is again distinctly heard, and now also another sound is heard which is produced by the normal blood current in vessels in or near the ear. In other words, we have blocked the natural outward pathway of sound conduction, with the result that vibrations normally imperceptible pass by bone conduction to the perceptive mechanism and are appreciated as sound.

It would seem that this theory of the causation of tinnitus might be found to apply to certain sounds for which other causes are sometimes assigned. For instance, it has frequently been noted, that, when the eyes are tightly closed by strongly contracting the orbicularis palpebrarum muscles, a low, buzzing sound is heard. This is supposed to be due to a sympathetic contraction of the stapedius, and is commonly spoken of as a "muscle sound." But if we will produce this sound by forcibly closing the eyes, and then compare it with the sound produced by placing a finger lightly in each external auditory meatus, we shall find that the two sounds are very similar in quality and pitch, though the latter is of greater inten-

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[1] Von Tröltsch: cited by Panse, Arch. of Otol., vol. xxviii, p. 365.

sity. May not this so-called muscle sound be thought of more correctly as a *conduction sound,* and as due to a temporary interference with outward sound conduction, during the contraction—which lasts but a few moments—of the stapedius muscle?

Since tympanic lesions may be accompanied by a high degree of congestion and, by pressing the stapes inward, may produce changes of the intra-labyrinthine pressure, it is evident that all cases of tympanic tinnitus do not necessarily belong to this class. That is, there may be other causative factors at work.

The outward conduction of sound may be interfered with by anything which occludes the meatus,—*e.g.*, cerumen, foreign body, furuncle, etc.,—or by anything which interferes with the mobility of the ossicular chain,—*e.g.*, tubal catarrh with retraction of the drum membrane, tubotympanic congestion, fluid effusions within the tympanic cavity, constricting bands binding the ossicles together or to the tympanic walls, etc., etc.

Viewed in this way, it is quite logical to treat these conditions locally and expect the tinnitus to subside as the parts regain their normal condition.

Blood Sounds.—The so-called blood sounds may be of arterial or venous origin. They may be dependent upon anatomical anomalies and not upon any pathological condition, in which case they are not to be looked upon as a symptom of disease, and are often not amenable to treatment. The close relation, for example, of the *internal carotid artery* and the *internal jugular vein* to the tympanum is well known. The anterior wall of the tympanum is separated from the internal carotid by a thin bony plate only, and the tympanic floor is in relation with the jugular fossa, which lodges the bulb of the jugular vein. As an abnormal congenital condition the carotid canal may project into and encroach upon the cavity of the tympanum, and it is not difficult to conceive that any malposition in this direction might give rise to sounds which the patient could not fail to hear. In the tympanic floor, also, dehiscences may exist either as a congenital defect or as a result of an old necrotic process which has undergone resolution. Many such cases have been observed during surgical operations and in the course of post-mortem examinations. At a meeting of German naturalists many years ago, Professor Körner,[2] of Rostock, presented, among other specimens, a temporal bone showing a fissure in the tympanic wall of the carotid canal. Dr. Dench[3] has recorded the case of a young woman upon whom he performed the operation of ossiculectomy. After removal of the ossicles, "a bluish mass was seen in the floor of the tympanum, which proved to be the bulb of the jugular vein, its exposure being due to a defect in the tympanic floor."

These cases are not unique, and are cited simply to emphasize the fact that tinnitus is not necessarily an evidence of disease. They corroborate the statement of Schwartze,[4] that "continuous noises may occur

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[2] Körner: Arch. of Otol., vol. xxxi, p. 119.
[3] Dench: Arch. of Otol., vol. xxvii, p. 297.
[4] Schwartze: cited by Panse, Arch. of Otol., vol. xxviii, p. 354.

during a lifetime in persons of normal hearing, due probably to anomalies in the course of vessels."

A persistent form of tinnitus, which we may also assume to indicate circulatory derangement, is that which may accompany visceral disturbances in other parts of the body. It is a well-recognized fact that visceral disorders—notably disorders of the pelvic and abdominal viscera—frequently produce aural symptoms. Woakes[5] explains this on the hypothesis of a nervous relationship between the viscera in question and the labyrinth. He points out that the nerves regulating the calibre of the vertebral arteries, and also of the basilar artery and its branches, including the internal auditory which supplies the labyrinth, come from the inferior cervical ganglion of the sympathetic. The stomach and other abdominal viscera are largely supplied by the pneumogastric nerves, and the communication between the vagus and the inferior cervical ganglion is established by means of a fasciculus from the vagus to the ganglion in question. Thus, in certain disorders of the stomach, for instance, impulses may be sent by way of the inferior cervical ganglion to the labyrinth, whereby its arteries are caused to dilate. Woakes claims that under such conditions the blood supply to the area involved "may be ten times as great as" under normal conditions. With this theory in mind it is no tax upon our credulity to accept the statement that "cases of tinnitus from constipation, subacute gastritis, and pathological conditions within the pelvis are of common occurrence."

In such cases it would be logical to expect the tinnitus to yield to treatment directed against the existing visceral disorders.

Patients with chronic endocarditis form another class who occasionally suffer with tinnitus. When with chronic valvular disease a murmur heard objectively at the base of the heart is transmitted to the vessels of the neck, an accompanying tinnitus may be clearly due to transmission of the cardiac murmur. Strumpel,[6] in writing of aortic regurgitation, speaks of cases in which, "by applying a stethoscope lightly over the femoral, the brachial, and often over the radial and ulnar arteries, a marked valvular sound" is heard. It would be surprising if such a murmur were not transmitted also to the patient's organ of hearing. A peculiarity of this variety of tinnitus is the fact that in some cases the sound heard by the patient may be heard also by the physician through the otoscope. As illustrating this statement may be mentioned the case of Spirig,[7] cited by Panse. His patient, a sufferer from aortic regurgitation, was troubled by subjective noises, at first pulsating, later becoming continuous. Objectively it was heard through the otoscope on the right side, a rhythmic, blowing sound; and a similar sound was heard through the stethoscope over the aorta, right carotid, subclavian, and brachial arteries.

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[5] Woakes: Deafness, Giddiness, and Noises, p. 135.
[6] Strumpel: System of Medicine, p. 289.
[7] Spirig: cited by Panse, Arch. of Otol., vol. xxviii, p. 371.

Another variety of subjective blood sounds which in rare cases may be heard by the physician through the otoscope is that which occurs with intracranial aneurisms. The basilar and internal auditory arteries are the vessels said to be most often affected. Excluding, then, a transmitted cardiac murmur, a pulsating noise synchronous with the pulse, heard subjectively and objectively in both ears, would suggest aneurism of the basilar artery; if heard only in one ear, the inference would be of aneurism of the corresponding internal auditory artery. Obviously, in such cases the tinnitus is not amenable to treatment except in so far as cardiac and nerve sedatives may lessen its intensity.

Blood sounds are usually of low or medium pitch, and are diagnosticated by their character and by the exclusion of other causes. Pulsating noises are of arterial origin, whereas venous sounds are uniform or non-pulsating in character.

There should also be mentioned a *venous blood sound*, which sometimes occurs with chronic or long-continued anæmia. It is a low, continuous hum, supposed to be due to the altered condition of the blood in its passage from the sinus into the bulb of the jugular vein. This sound is probably somewhat analogous etiologically to the hæmic bruit which in anæmia is sometimes heard objectively over the large veins of the neck, the so-called *bruit du diable.* The diagnosis depends upon the character of the sound, the absence of tympanic disease, exclusion by functional tests of labyrinthine disease, and evidences of anæmia as shown by the general symptoms and examination of the blood. Obviously the indications would be for iron, arsenic, tonics, or any treatment looking to the restoration of the blood to its normal condition.

Labyrinthine Sounds.—That subjective noises are invariably present at the onset of acute suppurative labyrinthitis is a fact universally recognized. This, however, is a class of cases in which by comparison with other more distressing phenomena, the tinnitus becomes a minor symptom. The subjective noises accompanying suppurative labyrinthitis will not be considered in this chapter.

There are, however, many cases of chronic non-suppurative middle-ear disease in which the accompanying tinnitus seems hardly to be explained as resulting from the tympanic changes alone. There are, for example, cases in which every known local therapeutic measure may be employed without in the slightest degree influencing the subjective noises.

In our study of such cases, we must remember that the auditory nerve not only receives and transmits sound impressions in response to sonorous vibrations (its normal function), but may also convey noise impulses to the auditory centres purely as a result of irritation or disturbance of its terminal cochlear fibres (perversion of function).

Many investigators (Grant,[8] Politzer,[9] Gruber[10]) have placed both

[8] Grant, Dundas: The Clinical Journal (London), vol. ix, pp. 241–7.
[9] Politzer: Diseases of the Ear, pp. 622–5.
[10] Gruber: Diseases of the Ear, p. 514.

congestion and anæmia of the labyrinth among the causes of tinnitus. George R. Field[11] over thirty years ago emphasized his conclusion that "slight variations, either of increase or diminution, in the pressure on the delicate structures (of the labyrinth) may give rise to severe tinnitus." There is much clinical and some experimental support of this view. Animals killed after frequently repeated doses of quinine, salicylic acid, etc., have shown marked labyrinthine congestion,—congestion sufficiently pronounced to have markedly increased intra-labyrinthine pressure; and these drugs in repeated doses usually cause distressing tinnitus in man. Amyl nitrite is another drug which invariably causes loud tinnitus, and here the effect so rapidly follows the cause that it seems possible to explain it only by reference to the increase in intra-labyrinthine pressure which the drug undoubtedly induces. On the other hand, after severe hemorrhage, when the patient lies in a condition bordering on collapse from a withdrawal of blood from all the tissues of the body, tinnitus is usually a prominent symptom, and in this case we are obliged to assume a reduction of intra-labyrinthine pressure.

Granting, now, that congestive conditions of the middle ear may cause vascular disturbances within the labyrinth, we can easily conceive that the commoner hypertrophic, or hyperæmic, form of chronic catarrhal otitis media may, through anastomotic channels, cause and maintain a constant state of moderate labyrinthine congestion.

It is a fact long known to pathologists that low grades of hyperæmia, long continued, almost invariably give rise to morbid changes in the tissues involved. It is probable, therefore, that in cases of obstinate tinnitus apparently dependent upon chronic catarrhal otitis media, we have also to contend with certain structural changes within the labyrinth which, being beyond our reach, account in many cases for our failure to relieve the subjective sounds. I believe that in this form of tinnitus more can be accomplished by regulating the patient's mode and habits of life, attention to hygiene, and possibly by constitutional remedies, than by local treatment.

Neurotic Sounds.—Those subjective noises may be classed as neurotic sounds which depend upon a hypersensitive condition of the auditory nerves. The tinnitus in most of these cases is to be regarded as a pure neurosis,—*i.e.*, it is dependent upon a functional disorder of the nervous system in which the auditory nerves share and sympathize. They may present no evidence of organic disease, yet are shown to be in a condition of increased or abnormal irritability.

This condition of acoustic hyperæsthesia may be produced by toxic matters circulating in the blood, which act as irritants upon the auditory nerves. In this way may be explained the temporary tinnitus occurring in some cases of chronic Bright's disease, also the tinnitus occurring occasionally without evidence of tympanic disease in the course of acute infectious diseases.

[11] Field: Medical Times and Gazette, June, 1878, p. 616.

The most characteristic cases of *neurotic tinnitus*, however, are found in patients suffering from neurasthenia or nervous exhaustion. In the diagnosis of these cases the process of exclusion naturally plays an important part. Thus, on examination of the patient, there may be found no disorder of the general circulation, no evidences of disturbed labyrinthine pressure, no visceral disorders which might affect the ear reflexly. Examination of the ear may reveal nothing abnormal within the meatus or tympanum, or, as is more common, a tympanic lesion being present, its correction does not result in relief of the tinnitus. Such negative results should suggest, at least, the possibility of a functional nervous disorder.

Careful questioning brings out the fact that the noise is made worse by bodily or mental fatigue; thus, it may be absent or hardly noticeable in the morning, after a night's rest, but reappears or becomes distressing as the day advances.

On functional examination, the hearing may at first seem nearly or quite normal. The tone limits are well maintained; lower tone limit normal, upper tone limit unchanged, or may be raised. For certain sounds—usually the higher notes of the musical scale, and such sharp sounds as are produced by the watch and acoumeter—the hearing may be abnormally acute,—*i.e.*, those sounds may be perceived further than the normal hearing distance.

But while the patient's hearing power may be normal, or even hyperacute at certain times, it will usually be found to show great variations, and to suffer rapid diminution as the patient becomes fatigued. Thus, the hearing power may be very much better in the morning than in the late afternoon after the fatigues of the day.

It is also characteristic of these cases (Dench[12]) that the patient may hear very well when conversing with one person, but with great difficulty when engaged in general conversation, the mental effort necessary to this attempt resulting in fatigue, which quickly reacts upon the auditory nerves.

Dundas Grant[13] has recorded some interesting cases of tinnitus due to nervous exhaustion. Having excluded tympanic lesions and the more usual constitutional causes, he made careful inquiry into the patient's habits, and in many cases elicited a history of overwork, prolonged anxiety, alcoholism, dissipation, sexual excess, or of some mode of life logically leading to nervous breakdown. Following up this clue, he found on functional examination marked hyperacusis,—a vibrating tuning-fork held opposite the patient's ear being heard twice the normal period of time, and the watch-tick being heard in some cases "across the room." In many of these cases the tinnitus was relieved only by regulation of the patient's life and by treatment appropriate to his nervous disorder.

Dana[14] places neurasthenia among the chief factors in the causation of

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[12] Dench: Diseases of the Ear, p. 605.
[13] Grant: Loc. cit.
[14] Dana: Nervous Diseases, p. 188.

tinnitus. He defines neurasthenia as a "functional nervous disorder, which is characterized by an excessive nervous weakness and nervous irritability, so that the patient is exhausted by slight causes and reacts to slight irritation."

With this definition in mind, it becomes evident that the aural symptoms are the logical results of the disease; that the tinnitus and hyperacusis are due to the "nervous irritability," while the variations of the hearing power under the influence of fatigue are due to the "nervous weakness."

It is probable that there are many cases of tinnitus of mixed origin in which the neurotic element is masked by other causative elements more on the surface; *e.g.*, a tympanic lesion, tubal catarrh, etc. It is the opinion of the writer that cases of tinnitus belonging, at least partially, to this class are far more common, even among the laboring classes, than is usually supposed. For, while neurasthenia is commonly supposed to be a disease to which brain workers and so-called upper classes are particularly prone, we have Dr. Dana's authority for the statement that sufferers among laborers and artisans are by no means uncommon.

Cerebral sounds, or sounds due to irritation of the auditory centres in the cerebral cortex, may be dealt with briefly.

This form of tinnitus is not very common in otological practice, and is of interest as much to the neurologist as to the otologist. Most authorities agree that complex or elaborated sounds—*i.e.*, sounds taking the form of voices, tunes, distinct words or sentences—are to be regarded as of cerebral origin.

The subjective noises which epileptics experience as a warning of impending attacks must also be regarded as due to a central lesion; and it is perhaps important to recognize this premonitory symptom from the fact that its presence is thought to bear somewhat on the prognosis of epilepsy, and to be of ill omen. Gowers[15] refers to the fact that "epileptics with such an aura are in greater danger than others of becoming insane." The hearing of complex or elaborated sounds is also believed by many to carry with it an unfavorable prognosis. While it is generally believed that the hearing of elaborated sounds points to irritation of the cerebral auditory centres, there are others who hold further that this symptom is an evidence of mental instability. Thus, Gruber[16] states that "patients who hear human voices, words, conversations, etc., are either mentally diseased or become so later." It seems hardly justifiable, therefore, to regard such subjective phenomena as aural symptoms, and as otological text-books and literature throw little, if any, light on the proper method of treatment, it would seem to be for the best interests of such patients to refer them to a competent neurologist.

In trying to differentiate the various forms of tinnitus in accordance

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[15] Gowers: British Medical Journal, Nov. 14, 1896.

[16] Gruber: Diseases of the Ear, p. 547.

with the underlying causes, we have the following sources of information to draw from:

1. Evidences of disease in any portion of the conducting apparatus as shown by physical examination.

2. History of the case, and character of the sound, as described by the patient.

3. Results elicited by careful functional examination.

4. Evidences of disease in other parts of the body, particularly as to the presence of digestive disorders, circulatory disturbances, blood dyscrasias, or disease of the nervous system.

5. Effect of certain drugs, either in relieving or aggravating the tinnitus.

The importance of a careful physical examination of the ear becomes evident from the fact that any appreciable lesion in any portion of the conducting apparatus must act at least as a contributing cause in the production of tinnitus, whatever the character of the sound and whatever the chief factor in its causation may be.

CHAPTER VII.

ACUTE INFLAMMATORY DISEASES OF THE EUSTACHIAN TUBE, MIDDLE EAR, AND MASTOID PROCESS.

Acute Tubal Catarrh; Eustachian Catarrh; Tubotympanic Congestion.—Tubal catarrh is the precursor of almost every form of tympanic disease, and as such deserves a more prominent place than is usually accorded it among diseases of the sound-conducting apparatus. It may be defined as an acute inflammation of the Eustachian mucosa, which may or may not give rise to aural symptoms, according to whether the lumen of the canal is, or is not, occluded thereby.

Etiology.—Among predisposing causes must be included all conditions interfering with normal nasal respiration. The presence of post-nasal adenoids is, therefore, a particularly potent factor, and children are much more frequently sufferers from tubal congestion and inflammation than is generally recognized. Among adults such obstructive lesions as hypertrophic rhinitis, septal deflections, ecchondroses and exostoses of the septum, etc., are conditions which are apt to induce recurrent attacks of Eustachian catarrh. Of exciting causes by far the most frequent and important is the common "cold in the head," or acute rhinitis, with which some degree of Eustachian congestion or inflammation is probably always associated.

Pathology.[1]—In a first attack of acute tubal inflammation the lesion is probably in most cases confined to the membrano-cartilaginous, or pharyngeal, portion of the canal. Beginning as a simple venous congestion, the condition is later converted into an acute exudative inflammation. In the great majority of cases it is probable that the lesion never advances to the point of actual tubal obstruction, in which case the tubal functions may not be seriously disturbed, and aural symptoms are slight or absent. The tube may, however, be occluded in one of two ways,—viz. (a) inflammatory swelling of the parts about the pharyngeal orifice may by pressure close the canal at this point, or (b) the inflammatory changes may extend into the osseous portion of the canal, obliterating its lumen at the isthmus.

Tympanic Changes.—So long as the Eustachian canal remains patent, or, rather, so long as it continues to perform its function of supplying air to the tympanic cavity, no changes are observed in the drum membrane. As soon, however, as the tubal function is disturbed,—*i.e.*, when the supply of air to the tympanum is diminished,—the air pressure within the middle-ear cavity becomes negative, and more or less congestion of the tympanic

[1] Our knowledge of the morbid changes characterizing this lesion is based largely upon conditions actually demonstrable during the onset and height of the attack, and their analogy with similar processes elsewhere in the body.

vessels ensues. This probably explains the prodromal symptom,—the feeling of occlusion or "stuffiness in the ear," which is sometimes complained of. When finally the Eustachian canal is completely occluded, the drum membrane is forced inward toward the inner tympanic wall by the unopposed atmospheric pressure from without. Inspection of the ear may now reveal the picture of a drum membrane showing no color or structural changes, *but greatly retracted.* It is probably at this point that the onset of aural symptoms is usually experienced.

Unless the tubal lesion is promptly controlled, very marked congestion of the tympanic mucous membrane may occur, and the peripheral plexus of veins along the margins of the drum membrane and the attachment of the hammer handle will be greatly engorged. This condition is described in some text-books as a distinct lesion under the name of *tubotympanic congestion.* It in reality constitutes a later stage of an acute tubal inflammation, and is a logical result of the lesion when unusually severe and prolonged.

Symptoms.—As before stated, complete absence of aural symptoms is by no means inconsistent with moderate grades of tubal catarrh. It is probable, therefore, that most of us are occasional sufferers from this lesion without experiencing symptoms referable to the ear. When occlusion of the canal gives rise to tympanic disturbance, the symptoms usually present themselves somewhat in the following order:

In a certain proportion of cases the attack is ushered in by a feeling of occlusion of the ear. This rather indefinite symptom is variously described by patients as a sensation of "stuffiness," or closure of the ear, or of the presence of a foreign body in the canal. This may persist for a few days and then disappear as the canal regains its normal condition; or it may be the forerunner of more definite symptoms of functional disturbance.

More commonly, according to the writer's experience, the first symptom complained of is that of marked impairment of hearing. This is often the more noticeable and distressing from its sudden development. The deafness is of the type characteristic of disease in any part of the conducting apparatus. Functional tests show diminished hearing for the watch, acoumeter, and for the conversational voice and whisper. Hearing for the lower musical tones is noticeably lessened, the upper tone limit not being disturbed. Hearing by bone conduction is always increased.

With the impairment of hearing the patient usually experiences rather loud subjective sounds, or tinnitus aurium. The noise usually is of high pitch, and is variously compared to the sound of a cricket, the escaping of steam, the noise of a shell against the ear, the sound of the surf, the ringing of bells, etc., etc. Quite often there is evident difficulty in recalling any natural sound with which to compare it.

In exceptional cases there is slight subjective vertigo, usually lasting but a few seconds, and rarely or never sufficiently pronounced to disturb static equilibrium. This symptom, when present, is presumably due to disturbance of intra-labyrinthine pressure, brought about through retrac-

tion of the drum membrane and inward pressure upon the ossicular chain and foot-plate of the stapes.

Aural pain is usually absent, practically always so during the first stage of tympanic disturbance from tubal catarrh. There are no constitutional symptoms,—*e.g.*, fever, pulse changes, etc.,—unless such are present as a result of intercurrent disorder, or of the disease to which the tubal catarrh is secondary,—*e.g.*, acute rhinopharyngitis, tonsillitis, grippe, etc.

Physical Signs.—Following the onset of aural symptoms, inspection by reflected light usually shows a drum membrane which is more or less retracted but otherwise normal. Since one of the most difficult tasks for the beginner is the detection of moderate degrees of retraction, it may be well to speak here of the changes in its physical appearance which usually characterize this condition. Extreme retraction, such as occasionally occurs in the presence of marked atrophy and loss of tension of the membrana tensa, is characterized by such obvious ballooning of the membrane inward and away from its points of attachment as to be easily recognizable even by the beginner. This, however, is an exceptional condition, such extreme displacement of the whole membrane being ordinarily prevented by the resistance which its inherent strength offers to the atmospheric pressure from without. It is quite important, therefore, that one should be able to recognize the moderate grades of retraction which are the more usual result of partial occlusion of the tube.

Cardinal Signs of Retraction.—Hammer Handle.—It will be remembered that the drum membrane is obliquely placed at the fundus of the bony canal, its outer surface looking outward and also strongly downward and forward. When yielding to pressure from without, it moves inward, backward, and upward, carrying the hammer handle with it. As seen through the canal, the hammer handle appears shorter and having a more decidedly backward direction than normally. This foreshortening of the hammer handle is in some cases extremely pronounced, and is one of the characteristic signs of a retracted drum membrane (Fig. 94).

There are certain cases, however, in which the distance between the umbo and the promontory is so small that the hammer handle is prevented from any extensive inward displacement. Under such conditions, while occupying very nearly its normal position, it is apt to appear abnormally broad and prominent from the fact that the drum membrane in moving inward is more or less folded about it (Fig. 96).

Processus Brevis.—The short process projects from just below the neck of the malleus in a direction both outward and upward. It is held firmly in position by the anterior and posterior ligaments (axis band), which are attached to the malleus a little below the level of the upper surface or point of the short process. In retraction of the drum membrane, the hammer handle moves inward, while the short process rotates outward. The short process appears, therefore, more prominent, as it really is, than when the membrana tensa occupies its normal position. Unusual prom-

inence of the short process constitutes a second sign of retraction (Figs. 94 and 95).

Light Reflex.—It will be recalled that this phenomenon of the normal drum membrane is due not to any structural change at this point, but simply to the fact that the drum membrane within this particular space falls into a plane which reflects the light directly upon the eye of the examiner. It is obvious, therefore, that with any change in the position of the lower part of the tense membrane, either the light reflex must be lost or its form changed. It may appear as a single, minute, shining spot immediately in front of the umbo (Fig. 94), or may be divided into two or more points of light. A common type of divided light reflex resulting from

Fig. 94. — Retracted drum membrane.

Fig. 95. — Retracted drum membrane.

Fig. 96. — Retracted drum membrane.

retraction occurs as a minute point of light in front of the umbo, and a second crescentic reflex lying near and parallel with the lower anterior arc of the annulus tendinosus (Fig. 95).

Another characteristic sign of retraction, which may be difficult to recognize in slight degrees of retraction, but is easily noticeable in cases of marked displacement, is due to the fact that the drum membrane is drawn away from the structures to which it is attached, causing them to appear unusually prominent. Thus, the annulus tympanicus, the anterior and posterior folds and the hammer handle, when this structure is not greatly foreshortened, stand out in exaggerated prominence. This condition is fairly well indicated in Fig. 96.

To epitomize: the cardinal signs of a retracted drum membrane are:

1. (a) Foreshortening and rotation backward of the manubrium mallei, or (b) apparent thickening and undue prominence of the same.

2. Unusual prominence of the short process.

3. Absence, or changes in the form, of the light reflex.

4. The anterior and posterior folds and the annulus tendinosus appear unusually prominent.

The Course of the Disease.—According to the development of tympanic changes, the disease may follow one of three roads toward final recovery.

1. In favorable cases the symptoms may last but a few days, disap-

pearing completely as the tubal inflammation subsides and the canal regains its normal calibre.

2. In cases of delayed resolution, the negative air pressure within the middle-ear cavity may result in very marked congestion of the mucous membrane lining the tympanum. This condition is usually characterized by engorgement of the venous plexus which is situated along the periphery of the drum membrane and along the margins of the hammer handle. The drum membrane, therefore, exhibits a line of redness in these regions,—*i.e.*, along the edges of the drum membrane and hammer handle. This marginal redness varies in intensity in different cases, but always preserves the characteristic of being differentiated by a distinct line of demarcation from the central area of normal membrane (Fig. 97). The symptoms are those described as belonging to the earlier stage of tubal catarrh, with the difference that aural pain (earache) may be present. It may, however, be absent, and when present is not usually very severe. This condition is spoken of by some authors as "tubotympanic congestion," a very useful and descriptive name, which should not, however, lead us into the error of regarding it as a separate lesion. The tympanic condition and accompanying symptoms usually subside as the tubal inflammation undergoes resolution.

Fig. 97. — Tubo-tympanic congestion.

3. There is still a third possibility in the conversion of the tympanic congestion into a simple acute inflammation with effusion of serum into the middle-ear cavity. This constitutes the purest form of acute catarrhal otitis media, as distinguished from acute purulent otitis media.

Prognosis.—Acute tubal catarrh—*i.e.*, in the first few attacks—tends to spontaneous and complete recovery. Frequent recurrence of tubal inflammation gives rise to chronic catarrhal conditions within the middle-ear cavity, leading to impairment of hearing later in life.

Treatment.—The treatment of tubal catarrh may be directed along three lines—viz., (a) internal and local remedies intended to relieve nasopharyngeal congestion, (b) local treatment of the tube aiming to re-establish its function, and (c) correction of nasal and nasopharyngeal lesions which might act as influences toward recurrence.

Treatment of the Nasopharynx.—In the first place, if the tubal lesion is very acute and accompanied by pronounced symptoms of nasopharyngeal congestion,—*i.e.*, those of a common "cold,"—the logical treatment is by active measures to combat the latter condition. Therefore, confining the patient to bed, brisk catharsis, and repeated small doses of quinine will do much to shorten the attack.

Even when it is not practical to carry out these measures, or when the symptoms do not seem to warrant them, much can be accomplished by the internal administration of appropriate drugs. Thus, quinine in grain

doses repeated every three hours during the day, or three times daily, may aid materially in shortening the attack. Personally the writer believes in the use of atropine or belladonna in acute tubal catarrh, their value being clearly traceable to their blennostatic action upon the nasopharyngeal and tubal mucous membrane. Belladonna may be prescribed with advantage in the form of the well-known "rhinitis tablets," Lincoln's formula,[2] half strength; or the extract of belladonna in gr. 1/8 doses may be combined with any of the coal-tar preparations. When the patient, in addition to characteristic tubal symptoms, exhibits the malaise and constitutional depression suggestive of grippe, or marked evidences of acute rhinopharyngitis, the following prescription will be found of great value,—particularly if the patient can be confined to bed for a few days.

℞ Capsules or powders, each to contain:
Extract. belladonnæ, gr. 1/8;
Phenacetini,
Salol, āā gr. iiss.
Sig.—One capsule (or powder) every four hours.

As with acute rhinitis, acute tubal catarrh may often at the onset be aborted by the timely use of appropriate drugs. After it has progressed beyond a certain stage, it must run its course, and the best that may be hoped for from internal medication is that the attack may be shortened thereby.

Locally, spraying the nose and throat night and morning with a cleansing alkaline solution (*e.g.*, Dobell's solution,[3] glycothymolin, etc.) seems in some cases to exert a favorable influence. Alkalol is a proprietary preparation which answers this purpose nicely, and has the considerable advantage of being practically non-irritating.

Certain cases of pronounced and intractable nasopharyngeal congestion or inflammation will respond favorably to a few repetitions of the following treatment: The nose and nasopharynx are thoroughly sprayed with a 5 per cent. solution of argyrol. This is allowed to remain a few minutes, and then washed out by means of the postnasal syringe with a warm normal saline solution.

If the writer has dealt at some length with the treatment of the nasopharynx, it must be remembered that the tubal inflammation is usually but an extension of a nasopharyngeal catarrh, and that whatever relieves the latter will provide the most logical treatment for the tubal lesion.

Inflation.—The first step in the direct treatment of the tube is by some

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[2] Lincoln's "rhinitis tablets," half strength, contain each: Extract. belladonnæ gr. 1/8, camphor. gr. 1/4, and quininæ sulphat. gr. 1/4.

[3] Dobell's solution:

℞ Acid. carbolici, gr. iv;
Sodii bicarbonat.
Sodii borat., āā gr xl;
Glycerini, oz. j;
Aquæ, q.s. ad oz. iv.

form of inflation. While this may be accomplished by the Politzer method, the catheter possesses the following considerable advantages: (1) We are able to inflate each ear singly and to regulate the pressure according to the resistance met with; (2) we obtain much more definite information as to the tubal condition from the clearer ausculatory signs (*i.e.*, through the otoscope); and (3) in our effort to force air through the more occluded tube, we are in no danger of unduly stretching the opposite drum membrane, as may easily occur in repeated Politzeration.

Local Anæsthesia.—The routine use of cocaine is not either wise or necessary for patients whose treatment requires repeated catheterization. There can be no doubt that repeated use of cocaine for any purpose carries with it some danger of establishing the drug habit. On the other hand, patients are often more or less nervous during a first examination, and it may then be wise to eliminate the discomfort incident to catheterization by applying a weak solution of cocaine. It should not be used in the form of a nasal spray, which anæsthetizes a larger surface than is necessary, and is more apt to cause reactionary engorgement of all the nasal tissues than when the application is limited to the pathway to be traversed by the catheter. This is easily accomplished by passing a cotton-wound applicator (Fig. 99) which has been dipped in a 4 per cent. solution of cocaine, through the inferior meatus of the nasal cavity corresponding to the ear to be inflated. The cotton should be passed slowly back and forth three or four times. A minute or two thereafter, it will be found that the catheter can be passed through the nose without causing any discomfort. After this first catheterization, the use of cocaine should be altogether omitted.

The technic of catheter inflation has already been described (pages 65–69), and need not be repeated here. It is a procedure which with care and skill can be made practically painless, or in careless or unpractised hands may become a veritable ordeal to the patient. The catheter should find its way easily and without application of force through the inferior meatus, the attempt to force it past any obstacle encountered resulting not only in pain, but in injury to the tissues. When an obstacle is met with in some nasal abnormality,—*e.g.*, a low septal ecchondrosis or exostosis,—it is usually possible, by slowly rotating the catheter, to find a pathway along which the catheter will enter the nasopharynx without force. When this is not possible, catheter inflation will be of no benefit, and we should either resort to the Politzer method or subject the patient to preliminary operative treatment for the correction of the nasal lesion. The latter alternative will generally afford the shortest road to a permanent cure.

Auscultatory Signs.—During inflation we may obtain important data as to the exact condition within the tubes from the sounds heard through the otoscope, or diagnostic tube. Thus, there may be (a) complete absence of sound characteristic of air entering the tympanum, this pointing to absolute occlusion of the canal; or (b) the presence of moist râles, changing in character, due to excessive secretion of mucus within the tube; or (c) the sound may be clearly indicative of air entering the tympanum, but

fainter and of higher pitch owing to the reduced calibre of the canal; or, finally, (d) there may be bubbling râles characteristic of air passing through fluid, indicating the presence of serum in the lower part of the tympanic cavity.

For all the above conditions, which are but stages or phases of the same lesion, the treatment is much the same. Inflation per catheter acts beneficially in several ways. Its first result is the re-establishment of the normal air pressure within the tympanum and the replacement of the drum membrane in its normal position. This frequently gives immediate relief of the symptoms, and in some cases restores the hearing almost to the normal standard. This improvement, however, is often of very short duration, the discomfort and functional disturbance recurring as the air in the middle-ear cavity is again exhausted. Inflation seems also to act directly upon the tubal mucosa, restoring its tone and aiding in the removal of mucus accumulations which are brought away with the return air current into the nasopharynx.

Inflation should at first be practised at short intervals,—*i.e.*, daily or on alternate days,—and later at longer intervals. The improvement under this measure is often very rapid and is clearly indicated by the progressive changes in the character of the sounds heard through the otoscope.

Local Application of Drugs.—There can be no doubt of the benefit in acute tubal catarrh from astringent applications to the pharyngeal mouth of the diseased tube. For this purpose the silver preparations give good results, — *i. e.*, nitrate of silver in solution of gr. 10 to 20 to the ounce, or argyrol in 25 per cent. solution. They are best applied by means of a cotton-wound applicator, the end of which is made to describe a curve similar to that of the Eustachian catheter (Fig. 98). This is passed through the inferior meatus of the nose and rotated into the pharyngeal mouth of the tube by exactly the same manipulations as are employed in placing the Eustachian catheter. In my experience argyrol is less irritating, and on the whole more effective, than the nitrate of silver. It can also be used more frequently,—*i.e.*, twice or three times a week, — in appropriate cases, while nitrate of silver, even in weak solutions, should not be applied oftener than once in seven or eight days. Another drug which is efficacious in some cases of Eustachian catarrh is the chloride of zinc in solution of gr. 10 or gr. 20 to the ounce.

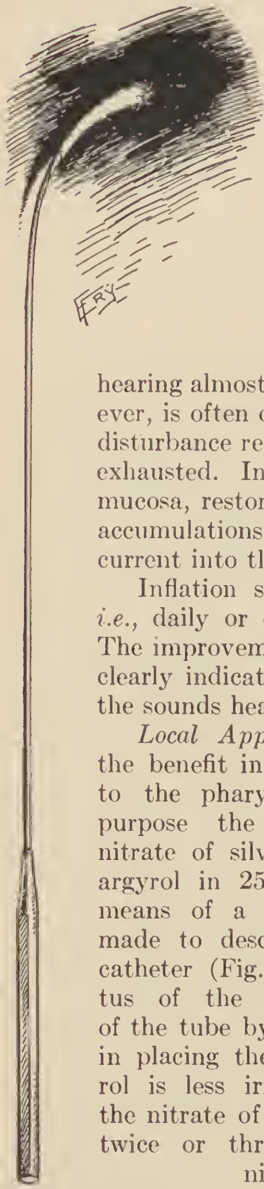

Fig. 98. — Eustachian cotton applicator.

Treatment of Tubotympanic Congestion.—When the lesion has reached the stage described as tubotympanic congestion (Fig. 97), the necessity for prompt therapeutic measures becomes urgent. Usually the treatment already outlined, but pushed rather more actively, will control the condition. Catheter inflation of the ear should be performed daily. The application of argyrol, in 25 per cent. solution, should be made to the nasopharynx and mouth of the tube on alternate days. Frequent gargling of the throat with alkaline or normal salt solution, as hot as patient can bear it, will in some cases act as a counter-irritant, relieving the tympanic congestion. Extract of belladonna, gr. ⅛, combined with small doses of quinine, repeated three times a day, or even four times daily, is of undoubted value in acute tubotympanic congestion. Earache, if severe, may be controlled by combining with the above an eighth of a grain of codeine for two or three doses. Finally, should the symptoms be aggravated by an effusion of serum within the tympanum, the best results will usually be obtained by a free incision through the posterior segment of the drum membrane. The incision is exactly the same as that to be described later under the treatment of acute catarrhal otitis media, and the subsequent cleansing of the canal must be carried out in the same way.

Correction of Nasopharyngeal Abnormalities. — The acute symptoms having subsided, it is of prime importance to the patient that the nose and nasopharynx should be carefully examined, and that any condition in this region interfering with nasal respiration or provocative of nasopharyngeal congestion should be corrected. Probably much could be accomplished by the correction of such lesions not only in preventing the recurrence of acute tubal attacks, but also in the larger field of preventing subsequent impairment of hearing,—*i.e.*, deafness in later years.

Tubal Catarrh in Children.—Before leaving this subject, the writer wishes to say a word as to the prevalence of tubal disease in young children.

Of all the causes of tubal inflammation none is so potent or so widespread in the number of its victims as the presence of pharyngeal adenoids. The author has long been convinced that retraction or congestion of the drum membranes constitutes one of the most constant and reliable physical signs of lymphoid hypertrophy in the nasopharynx; or, in other words, that the prevalence of tubotympanic disease in children is almost coequal with the prevalence of adenoids. If this view is correct, it is evident that the subject has not received due attention in otological literature and text-books, and, what is of more serious import, that the condition is more or less ignored or neglected by physicians and otologists alike.

There are two predisposing causes which render children particularly prone to tubal congestion,—viz. (1) the greater prevalence of adenoids, and (2) certain anatomical characteristics of the Eustachian canal in infancy and early childhood.

The more important anatomical differences between the infant and adult tubes are clearly indicated in Figs. 99 and 100. The heavy lines representing the Eustachian canals indicate their general direction in

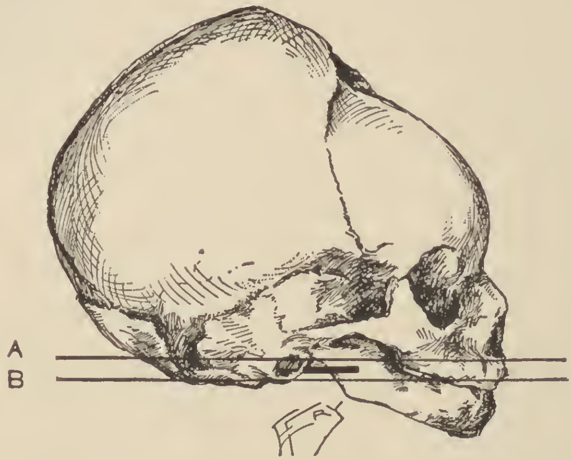

Fig. 99.—Skull of infant at term. Line A = level of nasal floor; line B = level of tympanic floor.

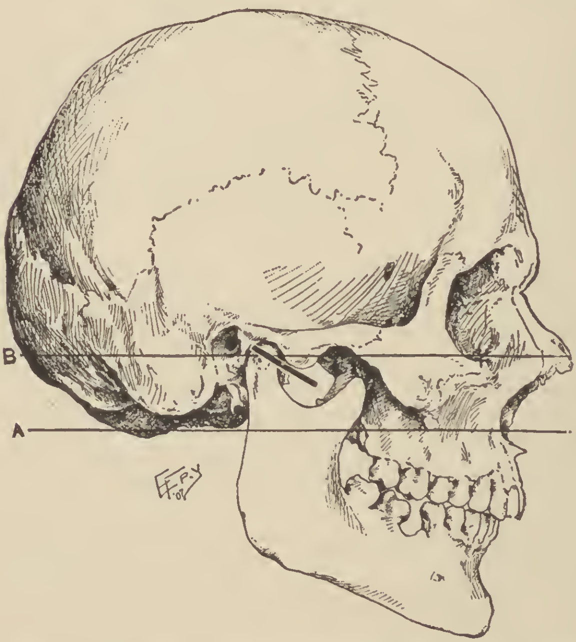

Fig. 100.—Skull of adult. Line A = level of nasal floor; line B = level of tympanic floor.

relation to the horizontal plane,—*i.e.*, they represent merely straight lines passing through the pharyngeal and tympanic orifices of the tube. The line A in each figure represents the level of the nasal floor, and the line B

the level of the tympanic floor. It is clear that the canal of the infant at term presents the following differences from the Eustachian tube of the adult:

(1) It is shorter (14 mm.) and horizontal in direction, whereas the adult tube is 33 to 38 mm. long, and passes obliquely upward and backward to reach the tympanum at a considerably higher level.

(2) The tympanic floor in the infant is on a level slightly below that of the nasal floor, while in the adult it is some 20 or 22 mm. above the floor of the nose.

(3) The pharyngeal mouth of the infant canal is on about the same level as the hard palate, whereas in the adult it is some 10 mm. above the hard palate.

To epitomize: The Eustachian tube of the infant is a short, relatively wide, horizontal canal, the pharyngeal orifice of which lies a little behind the choanæ and on a level slightly below that of the hard palate. Its physical characteristics seem, therefore, as compared with the adult tube, particularly favorable to the entrance of germs from the nasal secretion draining posteriorly into the pharynx, and to the development of vascular changes as a result of any pathological condition within the nose or nasopharynx.

Absence of Subjective Symptoms.—One reason why tubal catarrh in children is not oftener diagnosticated as such is the absence of such symptoms as a young child would be able to describe, or likely to complain of. Children of a certain age complain promptly of pain, but such indefinite symptoms as a sensation of fulness in the ears they probably soon grow tolerant of. We begin life with hearing power so far in excess of our actual need that a child's ears may be very seriously diseased before his hearing power becomes noticeably impaired. It is clear, therefore, that, if we are to recognize tubal catarrh in children at the stage at which it is most amenable to treatment, we must make our diagnosis chiefly by the physical signs.

Physical Examination.—I have already stated my belief that tubal catarrh is almost invariably present in children suffering from pharyngeal adenoids. Inspection of the ears of children in whom a physical examination has demonstrated the presence of adenoids will, therefore, usually show the drum membranes to be markedly retracted. When the adenoid growth is of such size as to interfere seriously with nasal respiration, the drum membranes are usually retracted to a degree rarely seen in adults. They may also be noticeably congested. Beyond this there may be little to attract attention to the fact that the Eustachian canals are diseased or are not properly performing their function.

Treatment.—The treatment is particularly important from the fact that the disease, if unchecked, probably represents the starting-point of morbid processes leading to deafness later in life. The indications are clear,—viz., removal of the pharyngeal growth, especial care being observed to avoid injury to the cartilage at the mouth of the tube, and subsequent treatment of the tubal lesion. For inflation of the ears we are usually obliged to resort to the Politzer method, few children being tolerant of catheter inflation. Aside from the method of inflation, the treatment is practically the same as for adult patients.

MYRINGITIS.

Acute circumscribed inflammations limited to the drum membrane have been described by Politzer, Schwartze, and other distinguished observers under the name of acute myringitis. Bezold,[4] on the other hand, felt so doubtful as to the propriety of separating such a lesion from a possibly coexisting inflammation of the middle ear, that he declined to recognize acute myringitis as a pathological entity. My own observations rather confirm the latter view, or at least convince me that the lesion is one of the rarest of aural diseases. I have seen, of course, innumerable cases in which the physical signs of inflammation were apparently confined to the drum membrane, but exceedingly few in which I was convinced that the middle ear was not also involved. I shall content myself, therefore, with a brief statement of the symptoms as described by others.

The condition is said to have been observed with comparative frequency in association with influenza. It may be ushered in by a feeling of fulness and discomfort in the ear. This usually gives way to lancinating ear pain of moderate severity. In some cases the earache is of very severe type. With the pain, loud subjective noises often add to the patient's distress. The hearing is usually only slightly impaired,—so little as often to escape the patient's attention. In the early stage inspection of the drumhead may show marked injection of the membrana tensa, which is often particularly pronounced in the region of the hammer handle. Later, with exaggeration of the above changes, one or more bullæ are seen, usually upon the posterior segment of the membrane. There may be also small ecchymoses, usually located in the posterior segment. The bullæ, or blebs, may rupture into the external auditory canal. The course of the affection is usually short, ending in recovery.

The disease is differentiated from acute otitis media by (a) the apparent limitation of the inflammatory changes to the external layers of the drum membrane, (b) the absence of bulging of the membrana tensa as a whole, and (c) the very slight impairment of hearing as compared with that usually accompanying acute middle-ear inflammation.

The treatment naturally varies with the severity of the attack. The milder cases probably recover with little or no treatment. Moderate pain may be relieved by the application of dry heat,—*i.e.*, lying with the affected ear resting upon a hot-water bag. In cases in which the earache is unbearably severe, the author believes that by far the safest method of obtaining relief is that employed in acute otitis media,—viz., free incision of the drum membrane and subsequent cleansing of the canal by means which will be described in connection with the treatment of acute tympanic disease. This plan of treatment gives prompt relief of pain and at the same time insures against the retention of inflammatory products which may collect within the tympanum.

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[4] Bezold: Text-book of Otology, p. 123.

ACUTE MIDDLE-EAR INFLAMMATION.

Nomenclature: Significance of Names in Common Use. — Before discussing separately the acute tympanic lesions, we should endeavor to define as clearly as may be just what we intend to convey as to the character of the lesions by the common terms "acute catarrhal otitis media" and "acute purulent otitis media." If the question were considered solely from a bacteriological viewpoint, it would be clearly possible to obtain in every case a specimen of the tympanic secretion, and according to the absence or presence of bacteria to say that we have to deal with either a serous or a purulent otitis media. It is neither possible nor desirable, however, in actual practice to base one's diagnosis wholly upon the bacteriological findings, and there are obvious advantages in the adoption of a simple classification which may be based upon such data as one may obtain from a careful examination of the patient.

According to the investigations of Zaufal and many who have followed his line of work, there are reasonable grounds for questioning whether acute inflammatory processes ever involve the middle-ear cavity in the absence of pathogenic bacteria. He found in a large series of examinations that pus germs were always present in the tympanic secretion taken from acutely inflamed ears; and, *per contra*, that no cultures could be obtained from secretions taken from apparently normal ears. Accepting this theory as generally correct, we must yet admit that certain congestive conditions —*e.g.*, tubotympanic congestion with effusion of serum—may be accompanied by distention and vascular engorgement so pronounced as to reproduce all the clinical and physical phenomena of acute purulent otitis media and to constitute a condition not clinically distinguishable therefrom.

Bezold, in discussing the nomenclature of tympanic disease, objected to the use of the word "catarrhal" as applied to middle-ear inflammations. He based his objection largely upon certain histological features of the lining membrane of the tympanum which seemed characteristic of a serous, rather than of a mucous, membrane. Thus, while the mucous membrane of the cartilaginous portion of the Eustachian tube is thick, lined by several layers of ciliated epithelium, and contains many acinous glands, the lining membrane of the tympanum is exceedingly thin, contains no acinous glands, and is devoid of cilia except upon the floor. In the atrium it consists for the most part of a single layer of basement cells very closely adherent to the periosteum, and has a decidedly endothelial character. The mucous membrane of the pharyngeal half of the Eustachian canal secretes more or less rather thick mucus, whereas the normal secretion of the tympanum is very thin and serous in character. Bezold, therefore, proposed to omit the term "catarrhal" from descriptions of tympanic inflammation, and to substitute the word "simplex,"—thus, "otitis media simplex acuta" as differentiated from "otitis media catarrhalis acuta." The word "simplex," thus used, is in no way descriptive, however, and is open to the

further objection that it would prove in many cases actually incorrect or misleading, since it is often impossible to determine clinically whether an acute tympanic lesion is or is not purulent in character. It would seem better, therefore, not to discard old, time-honored names, which have obtained such widespread adoption, but rather to define clearly the character of the lesion they are intended to describe.

There are three common forms of acute middle-ear inflammation, an appreciation of which helps to a clearer understanding of the varying phases of tympanic disease,—viz. (1) *An acute serous otitis media,—i.e.* a non-purulent inflammation with effusion of serum into the cavity of the atrium; (2) *an acute purulent inflammation confined to the atrium;* and (3) *an acute purulent inflammation involving both the atrium and the tympanic vault.* It may be quite impossible, however, by its clinical manifestations to distinguish an acute serous, or non-purulent, otitis media from an acute purulent otitis media. In either the membrana tensa may be red and bulging, the subjective symptoms are often identical, and the indications for treatment are practically the same. From the standpoint of practical otology, therefore, it seems best to consider acute middle-ear inflammation under two heads,—viz. (1) "*Acute catarrhal otitis media,*" by which term we shall designate any acute inflammation, purulent or non-purulent, confined to the atrium; and (2) "*acute purulent otitis media,*" or an acute inflammation involving both the atrium and the tympanic vault. These two conditions undoubtedly represent respectively the simpler and the more serious forms of acute tympanic disease as met with in actual practice.

Otitis Media Catarrhalis Acuta; Acute Catarrhal Otitis Media.—These terms we apply to the simpler form of acute middle-ear inflammation, —*i.e.*, an acute inflammatory process confined to the atrium.

Etiology.—The causes have already been enumerated in a foregoing chapter. Depressed constitutional states, and therefore exhausting diseases, render the individual more prone to tympanic disease. Obstructive lesions of the nose or nasopharynx—*i.e.*, conditions interfering with nasal respiration—are strongly predisposing factors. Pharyngeal adenoids react injuriously upon the ears, and are responsible in large measure for the prevalence of tympanic disease among children.

Exciting Causes.—Gauging their importance as proportional to the number of cases to which they give rise, the exciting causes should be mentioned in the following order: (1) *Acute nasopharyngitis,—i.e.*, the common "cold in the head." More cases of acute middle-ear inflammation can be accounted for as secondary to this affection than can be traced to any other cause. (2) *The acute exanthemata:* scarlet fever, diphtheria, and measles cause acute tympanic disease with even greater frequency than is generally recognized by the medical profession. Scarlet fever is accompanied by acute tympanic disease more frequently than is diphtheria, and the proportion of scarlatinal cases which go on to acute suppurative mastoiditis is considerably larger. Measles is a very frequent cause of acute otitis media of rather severe type in which mastoid suppura-

tion occurs with marked frequency. Acute epidemic influenza gives rise to acute otitis media, often of very severe type. The proportion of cases in which this complication occurs varies in different epidemics according to variations in the average severity of the disease. (3) *The nasal douche:* it is a recognized fact that many cases of acute middle-ear inflammation are caused by the careless use of the nasal douche in cases of atrophic rhinitis, ozæna, etc. In the milder cases of nasopharyngeal disease, the nasal spray will be found an efficient, and certainly safer, method of cleansing the nose. (4) *Diving or swimming under water:* the frequency of tympanic inflammation from this cause is evidenced by the large number of patients presenting themselves each year at the beginning of the swimming season. The injury to the ears may occur in one of two ways,—viz. (a) by expulsion of water taken into the nose or throat through the Eustachian tubes or (b) by impact of the cold water against the drum membrane. The latter danger may be practically eliminated by stopping each auditory canal with a pleget of moistened absorbent cotton. (5) *Traumatic injuries of the drum membrane* are a comparatively rare cause of acute middle-ear inflammation. Of such injuries the commonest is that caused by a forcible box or slap upon the ear, the drum membrane being ruptured by the sudden condensation of the air in the external auditory meatus.

Micro-organisms found in the Ear during Acute Middle-ear Inflammation.—A very large number of micro-organisms have been found in the secretions taken from the ear during acute otitis media. Those most frequently found are the streptococcus, the pneumococcus, and the streptococcus mucosus. Next in frequency are the staphylococcus albus and staphylococcus aureus, and after these come the bacillus proteus, the bacillus pyocyaneus, and a long list of organisms which do not call for special mention here.

As regards the comparative virulence of these germs in relation to aural disease, there is no longer room for doubt that the streptococcus and pneumococcus give rise to a type of middle-ear suppuration which leads more frequently to intracranial infection than do the tympanic lesions caused by the staphylococci or other germs mentioned. Suepflé, from an analysis of the bacteriological findings in a large series of cases, concluded that cases of acute otitis media due to staphylococcus infection do not give rise to intracranial complications. While this view is extreme, and therefore incorrect, it serves to emphasize the infrequency of intracranial disease secondary to staphylococcus infection. The streptococcus, on the other hand, is unquestionably responsible for a majority of cases of intracranial disease of otitic origin.

Accepting the above statements as in the main correct, one must yet acknowledge that one can not prognosticate from the micro-organism found in the tympanic discharge the future course or severity of the aural disease, this being determined quite as much by the patient's systemic condition,—*i.e.*, the question of normal or lowered resistance. A staphylococcus may, therefore, produce clinically an exceedingly severe type of

inflammation; whereas a mild type of acute otitis media may result from a streptococcic invasion of the ear.

Anatomical Conditions in Relation to Symptoms.—It may be well, before discussing clinical phenomena, to review briefly certain anatomical features of the atrium which bear rather directly upon the symptoms and course of the simpler form of acute otitis media.

It will be recalled that the cavity of the atrium is lined by an exceedingly thin membrane very closely adherent to its walls. Externally the atrium is closed by the tense, inelastic and rather unyielding membrana tensa. Superiorly the atrium is more or less completely separated from the tympanic vault by the structures massed in this situation,—viz., the neck and short process of the malleus; the lower anterior extension of the body of the incus; the anterior, external, and posterior ligaments of the malleus, and the mucous membrane which surrounds them. From the anterior wall of the atrium the Eustachian canal leads forward, inward, and downward into the nasopharynx. Germs reaching the tympanum from the nasopharynx by way of the Eustachian tube lodge first, therefore, in the atrium.

An inflammatory process originating in and confined to the atrium probably represents changes taking place somewhat in the following order: (1) Marked dilatation and engorgement of the vessels of the lining membrane; (2) transudation of serum and migration of leucocytes from the veins into the tissues of the mucoperiosteal lining. The consequent inflammatory thickening of the lining membrane has the important mechanical effect of rendering the separation of the atrium from the vault more complete. The bony Eustachian tube is also more or less closed by inflammatory swelling in the region of the tympanic orifice. With the formation of pus or effusion of serum into the atrium, the walls of that cavity are, therefore, soon subjected to pressure, the outer wall, or membrana tensa, being the one which offers least effective resistance. Hence the early spontaneous rupture of the drum membrane—usually in six to twenty-four hours—which commonly occurs in acute catarrhal otitis media.

As compared with the vault, the relatively small amount of connective tissue in the atrium (consisting chiefly of its thin and closely adherent lining membrane) constitutes another condition favorably influencing the progress of the disease. That is to say, the fastigium being passed, the tissues more quickly regain an approximately normal condition, the patency of the Eustachian tube being restored, and the mechanical conditions favorable to resolution being more quickly established.

Symptoms and Signs.—When acute otitis media occurs as a complication of one of the acute infectious diseases, the otitic symptoms may for a time be more or less masked by those of the initial disease. When, on the other hand, the aural disease is not secondary to a systemic infection, the onset is usually sudden and clearly defined.

As a prodromal symptom, a sensation of fulness or occlusion of the ear is noted by some authors as of frequent occurrence. This symptom,

when present, is clearly a result of the tubal catarrh, or congestion, which almost invariably precedes or accompanies acute catarrhal otitis media. According to the writer's observation, the symptom first experienced, or at least first complained of, by the patient is usually pain.

Onset.—The earache is usually sudden in its development. Following a few premonitory twinges, it soon assumes the character of a constant pain, rapidly reaching an unbearable degree of severity. Not infrequently the patient retires at night with no noticeable discomfort, to be awakened during the night or toward morning by the severity of the ear pain. Once established, the pain is usually constant, but is subject to exacerbations of intensity. With the beginning of pus formation, the pain becomes throbbing or "drawing" in character, being sometimes compared by the sufferer to the severe form of toothache usually accompanying an abscess at the root of a carious tooth. Naturally, sleep becomes impossible; and when prompt relief is not forthcoming, the resulting insomnia adds to the nervous strain to which the patient is subjected. The severity of the pain often distracts the patient's attention from lesser symptoms. If questioned, however, he usually becomes conscious of subjective sounds,—*i.e.*, tinnitus aurium. The hearing is also noticeably impaired, showing certain characteristic changes which will be referred to later.

The constitutional symptoms vary widely with the age, and also to some extent with the nervous constitution of the patient. With adults, the temperature may throughout be normal or nearly so, or it may be moderately elevated. With infants and young children, on the other hand, even the mildest type of acute middle-ear inflammation is usually announced by rather high fever,—*i.e.*, temperature ranging from 102° to 105° F. The temperature variations which both adults and children occasionally exhibit with mild grades of tympanic inflammation depend probably upon individual differences in the stability of central nervous control. That is to say, it seems necessary in some cases to refer the fever to a reflex disturbance of the cerebral heat centres, rather than to the influence of septic absorption from the ear. Certainly the rapid and considerable elevations of temperature frequently seen in children suffering from the simplest type of acute tympanic inflammation are in many cases most easily explained in accordance with the above hypothesis,—*i.e.*, as a reflex phenomenon.

With patients of all ages, adults as well as children, digestive disturbances are common during the early stages of an acute catarrhal otitis media.

Spontaneous Rupture of the Drum Membrane.—As we have seen, the atrium during acute catarrhal otitis is a small, closed cavity, the outer, membranous wall of which represents the direction of least resistance. Obviously, the membrana tensa must give way before any rapid accumulation of pus or serum. Spontaneous rupture of the drum-head usually occurs within six to twenty-four hours after the onset,—or, to be more exact, after the appearance of the initial earache.

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With the rupture of the drum membrane and the appearance of aural discharge, there is usually complete cessation of pain. The adult patient usually experiences an immediate sense of relief, and the child, which has been tossing about the bed in distress, now falls into a sleep of exhaustion. The temperature, which in young children is usually elevated, may fall within a few hours to the normal line.

With adult patients, two symptoms, probably in some degree present from the onset, are now thrown into bolder relief, and become the patient's chief cause of concern,—viz., tinnitus aurium and impairment of hearing.

Tinnitus Aurium.—The subjective sounds apparently vary greatly in different individuals. On the other hand, it may be that the variations are often more apparent than real, depending upon the natural difficulties in describing a purely subjective sensation. Thus, we are frequently told that the noise is like the "noise of a tea-kettle," "the escaping of steam," "whistling of wind in the trees," "the sound of a cricket,"—all which comparisons may be intended to describe the same character of sound. Again the sound is compared to a ringing of bells or to a shrill whistle,—*i.e.*, sounds of different character. From the average statements of intelligent patients, one is led to believe that the sound is usually of rather high pitch.

Tinnitus which has made its appearance during, and as a result of, an attack of acute otitis media usually disappears when the last physical evidence of inflammation has gone. It is, however, in my experience often the very last symptom to disappear.

Impairment of Hearing.—The hearing is always impaired at the height of an acute catarrhal otitis media. At the onset,—*i.e.*, during the stage preceding the accumulation of fluid in the tympanic cavity—the impairment may be slight, or not noticeable. As serum or pus displaces the air in the tympanum, however, the hearing power is usually considerably reduced.

The type of impairment assumes to some extent the general character of deafness due to disease in any part of the conducting apparatus,—*i.e.*, hearing by bone conduction is increased, and the ratio between hearing by bone conduction and by air conduction is changed, though not necessarily reversed.

Bezold called attention to certain features of the disturbance of hearing as characteristic of acute middle-ear inflammation with effusion. In acute otitis media with bulging of the drum membrane, bone conduction is increased, and the tuning-fork held in contact with the mid-line of the skull is referred definitely to the diseased organ. With this increase in bone conduction there is, however, according to Bezold, surprisingly little disturbance of audition for the low musical tones, some of his patients hearing tones as low as 16 d.v. This is the reverse of what one usually finds in chronic lesions of the conducting mechanism, in which the lower tone limit is almost invariably considerably elevated. Bezold explains this difference by reference to differences in the pathology of the two types

of disease. Thus, in chronic middle-ear disease, either suppurative or non-suppurative, there is usually present either partial destruction of the drum membrane, or fixation of the membrane or ossicles. Either of these conditions would interfere markedly with the slow and extensive movements involved in the production of very low musical tones. In acute catarrhal otitis media, on the other hand, disturbance of function is due largely to the presence within the tympanum of fluid which acts as a weight upon the drum membrane and ossicles, interfering with the acuteness of hearing for all sounds, but not necessarily offering special resistance to the slow and relatively wide vibrations necessary to the transmission of the lower tones of the musical scale.

Fig. 101.


Fig. 102.


Fig. 103.

Figs. 101, 102, and 103.—Acute catarrhal otitis media. 1st, 2d, and 3d stages.

Obviously, the presence of fluid in the tympanum is not the only factor in the deafness of acute otitis media, the other being the inflammatory infiltration of the drum membrane and tympanic mucosa. This explains the fact that some degree of deafness often considerably outlasts the cessation of the discharge and final closure of the drum membrane.

Physical Changes in the Drum Membrane.—The physical appearance of the drum membrane varies materially with the time which has elapsed since the onset. If seen very shortly after the earache is first experienced, the drum-head will be found deeply injected,—*i.e.*, exceedingly red. When different portions of the tense membrane show variations in this respect, the upper part is usually more deeply injected than the lower portion, which may be nearly or quite normal in color. In other words, the injection is said to appear first above and extend downward; but there is never any line of demarcation between the injected and normal portions, the one shading gradually into the other. In a majority of cases, however, the whole membrana tensa when first seen by the physician is uniformly red (Fig. 101).

If the examination be delayed a few hours, inspection of the drum membrane may reveal an entirely different picture,—*i.e.*, the membrana tensa is now not only exceedingly red, but bulges markedly outward into the lumen of the meatus. A slight central depression may be seen in the position of the umbo. Other than this, the normal landmarks, including the short process and handle of the malleus, may be more or less obliterated or hidden from view (Fig. 102).

If the examination be still further delayed, we shall be likely to find the

canal filled with pus, in which case we can, of course, see nothing of the drum membrane until the ear has been irrigated or the meatus otherwise cleansed. It is then possible to locate the perforation, which is situated practically always in the posterior segment of the membrane. Most frequently it is found in the posterosuperior quadrant,—*i.e.*, behind the hammer handle. Occasionally a perforation is so minute that one is not quite sure of its recognition. In such a case, if the patient is required to perform Valsalva inflation while the drum membrane is kept in view, a drop of pus will be seen to exude, thus locating definitely the site of the perforation (Fig. 103).

The Discharge.—Following rupture or incision of the membrane, the discharge may vary considerably, in accordance with certain conditions, —*e.g.*, the constitutional condition of the patient, the character of the infection, the time elapsing between the onset of the disease and the rupture of the drum membrane, and, of course, with the efficiency of the treatment. If the inflammation is serous, or non-purulent, and the rupture of the drumhead has occurred early, the discharge may remain serous in character and last but a few days. If, on the other hand, the patient is in a constitutionally exhausted condition, or the infection is of virulent type, and particularly if the rupture of the drum membrane has been considerably delayed, the discharge is likely to be more profuse and to persist longer.

Taking as an example a case in which an intensely red and bulging drum membrane has been properly incised, the discharge may pursue somewhat the following course: Frequently it does not reach its maximum flow during the first twelve or twenty-four hours following the incision. The writer has repeatedly been impressed with the relatively moderate discharge immediately following myringotomy in cases of severe tympanic inflammation. After the first twelve or twenty-four hours it rapidly increases in amount, usually reaching the maximum on the second or third day. From this point it may for a few days be exceedingly profuse, then gradually receding. Its disappearance may be quite rapid, or, for no reason discernible in the physical character of the lesion or the type of infection, be comparatively slow. Finally, after the amount is reduced to an apparent minimum, it may persist obstinately for days, suddenly ceasing with the complete closure of the drum membrane. Following the closure of the incision, the drum membrane may rapidly regain its normal appearance or weeks may elapse before all traces of the recent inflammatory process have disappeared.

The above describes somewhat in detail the usual course of an attack of acute catarrhal otitis media. The whole process, from the appearance of the discharge to final closure of the drum membrane, may run its course in a week or less, or may be spread over two or three weeks or more. To understand these otherwise puzzling variations, we must recognize in the patient's general power of resistance, or departure from the normal therein, a factor quite as potent in deciding the course of the disease as the character of the infection itself.

In the writer's opinion, serious complications of acute catarrhal otitis media rarely occur without previous change in the character of the lesion,—*i.e.*, without extension of the infection to the structures of the vault.

The PROGNOSIS as to ultimate recovery is good. The drum membrane usually heals without noticeable cicatrices or structural changes. Permanent impairment of hearing is exceedingly rare. There can be no doubt that a mild attack in which final healing is unduly delayed is more apt to result in some permanent impairment of function than a severe lesion in which prompt local recovery occurs. On the other hand, a very prolonged attack may leave absolutely perfect hearing.

Acute Suppurative Otitis Media; Acute Purulent Otitis Media (Otitis Media Purulenta Acuta).—These terms are employed to describe a suppurative inflammation involving both atrium and vault. In many cases the inflammatory process seems to originate in the vault, spreading thence downward to the atrium; in others, an infection of the atrium extends upward to the attic.

ETIOLOGY.—The causes of acute purulent otitis media are the same as those giving rise to the simpler form of acute tympanic inflammation. They do not, therefore, call for repetition here. So also of the bacteriology,—there is no organism which may cause one grade of acute middle-ear inflammation and not the other. That is to say, the same exciting cause, or the same pathogenic micro-organism, may give rise either to the simpler or the more severe form of otitis media,—this depending perhaps to some extent upon variations in the individual's vitality or resistance power to disease.[5]

ANATOMICAL CONDITIONS INFLUENCING THE PATHOLOGY OF THE DISEASE.—The anatomico-surgical features of the vault are perhaps best shown by comparing them with those of the atrium. It will be remembered that the atrium contains but little connective tissue other than the thin and closely adherent membrane lining its walls. Inflammation tends, therefore, to be self-limited, by reason of the very limited field involved, and the rather unfavorable nidus provided for the growth of germs. The mechanical separation of the atrium from the vault is rendered more complete by the inflammatory swelling of mucosa, this favoring early rupture of the drum-head, with consequent drainage and relief of tension,—*i.e.*, early resolution. As congestion about the Eustachian orifice subsides, the Eustachian canal provides a supplementary drain for the escape of serum or pus.

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[5] The frequent occurrence of suppurative lesions, apparently originating in the vault, is rather difficult to explain. Why should germs presumably entering by way of the Eustachian canal primarily infect the vault? In trying to account for this phenomenon, the following theory has seemed to the writer the most satisfactory,—viz., that the atrium presents certain conditions unfavorable to the development of purulent inflammation which do not exist in the vault; that germs entering by way of the Eustachian canal may pass through the atrium without infecting its mucosa, and, reaching the vault, may find there the conditions favorable for the inception and spread of a suppurative inflammation.

The vault (Fig. 104) presents the following features bearing upon the pathology of suppurative lesions originating therein: (a) It is directly continuous posteriorly with the so-called mastoid antrum. Pus collecting from a primary infection in the vault is not, therefore, quickly subjected to pressure,—flowing rather backward along the pathway of least resistance into the antrum. *The antrum is early involved in acute purulent otitis media*, and from the onset the danger of suppurative mastoiditis is greater. The escape of pus downward into the atrium frequently represents a later stage,—*i.e.*, is subsequent to the accumulating of pus within the antrum. Spontaneous rupture of the drum membrane is often therefore delayed, usually from forty-eight hours to a week. (b) There is no natural path-

Fig. 104.—Diagrammatic picture of membranous partitions of vault.

way providing for the free escape of pus from the vault, and even extensive incision of the drum membrane does not provide free and adequate drainage from the antrum. (c) The lining membrane of the vault is not closely adherent as in the atrium, but hangs in folds from the tympanic roof, covering the head of the malleus and body of the incus, and dividing the attic into an outer and inner compartment. Another fold of mucous membrane is usually present, passing horizontally across the outer chamber of the vault, and dividing it further into an upper and lower space. This surplus of vascular connective tissue forms a suitable nidus for the growth of infective germs, and favors the development of suppurative inflammation. The subdivision of the vault cavity into membranous compartments is also distinctly favorable to the retention of pus, and therefore to the spread of infection.

Symptoms and Signs.—The subjective symptoms of acute suppurative otitis media do not differ greatly from those of the simpler form. Earache is usually the first purely aural symptom of which the patient complains.

The ear pain resulting from a primary infection of the vault is, perhaps, a little less sudden in its attack, and somewhat slower in reaching its maximum intensity, than that accompanying a similar lesion confined to the atrium. It is also much less likely to be relieved by early rupture of the drum membrane. It is described as a most unbearable type of pain,—throbbing or boring in character,—and soon reaching a degree of severity rendering sleep impossible. From the onset, the patient usually experiences subjective noises,—this symptom, however, being over-balanced by the severity of the pain, and therefore rarely complained of at this stage of the disease. The hearing may at first be but little disturbed, this depending upon the extension of the inflammation to the membrana tensa and structures about the oval window and incudostapedial joint, and upon the accumulation of pus within the atrium. As the lesion advances, the hearing is always markedly impaired.

Some degree of fever is usually present in the acute stage. With adults the temperature may rise to 102° F. or more, or may be but slightly above the normal. With infants and young children, on the other hand, rather high temperature is the rule. Owing partly to the fever, when present, but chiefly to the severity of the pain and the consequent insomnia, there are few circumscribed lesions which will more quickly exhaust the patient than an attack of acute middle-ear inflammation which has not been relieved by rupture or incision of the drum membrane.

Spontaneous Rupture of the Drum Membrane.—The interval between the initial pain and the rupture of the membrane is usually much longer than in the simpler form of acute otitis media. In my experience, it varies all the way from forty-eight hours to a week. This wide variation is probably explained by differences in the early pathways of extension. If, for example, a suppurative process in the vault extends quickly in the direction of the atrium, or is quickly followed by a secondary infection of the lower tympanic space, pus collecting there will be likely to cause a comparatively early rupture of the membrane. On the other hand, a primary infection of the vault, spreading chiefly backward in the direction of the antrum, may torture the patient for days before pus, accumulating in the atrium, causes rupture of the membrana tensa. Such cases are best studied in dispensary practice, where patients frequently come under observation who have suffered three, five, or even six days without rupture of the membrane. It is not difficult to appreciate why final rupture of the membrane in these cases often affords much less relief than that which almost invariably follows the early rupture of the drum-head in acute catarrhal otitis media.

Physical Signs.—It is clear that the physical changes in the drum membrane must vary with the progress of the disease. We shall try, therefore, to describe certain conditions, representing progressive stages of the lesion, in the order in which they would be likely to occur.

If the patient is seen very shortly after the initial symptom, we may find the following conditions present:

1. The membrana tensa throughout the greater part of its extent may

be quite normal in color and appearance. The upper posterior portion of the tense membrane,—that part immediately contiguous to Shrapnell's membrane,—and Shrapnell's membrane itself, are intensely red, and, if not actually bulging, have the appearance of acute inflammatory infiltration (Fig. 105). This condition is very frequently observed in patients who are seen just after the onset, and usually indicates a suppurative process in the vault, which later will extend downward and involve the atrium.

Fig. 105.

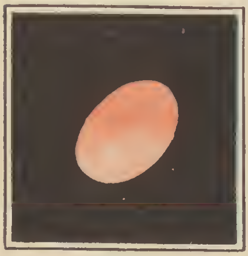

Fig. 106.

Fig. 107.

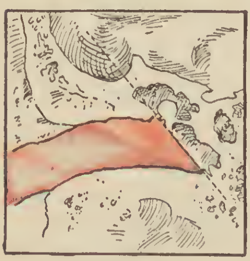

Fig. 108.

Figs. 105, 106, 107, and 108. —Acute purulent otitis media, 4 successive stages.

2. A few hours or one or two days later, we may see the following changes: the upper half or whole of the membrana tensa is red, or injected. The injection in the region of Shrapnell's membrane is, however, much more intense, and the inflammatory thickening (infiltration) in this region is so pronounced as to indicate clearly a suppurative process behind Shrapnell's membrane,—*i.e.*, in the vault (Fig. 106). This picture is often seen in acute suppurative inflammation of the attic, and occurs at a period following the onset which in a similar lesion of the atrium would have been marked by noticeable bulging or rupture of the drum membrane (Figs. 102 and 103).

3. The upper posterior part of the membrana tympani—*i.e.*, the part including and immediately adjacent to Shrapnell's membrane—is markedly bulging, clearly indicating the presence of pus in the tympanic vault (Fig. 107). With this condition, the entire membrana tensa also is invariably inflamed. The bulging, however, is confined to, or most conspicuous in, the region of Shrapnell's membrane.

4. The canal contains pus, removal of which reveals the following conditions,—viz., entire drum membrane (membrana tensa and membrana flaccida) red and bulging. Small perforation in posterior segment of membrana tensa. Posterosuperior canal wall—*i.e.*, that part immediately adjoining the drum membrane—is frequently very considerably swollen, so that no line of demarcation is here discernible between canal wall and drum membrane (Fig. 108). This condition always indicates a severe suppurative process within the vault, and frequently coexists with inflammation of the mastoid cells. It is commonly spoken of as "sagging of the posterosuperior canal wall."

Antrum Tenderness.—It must be clear, from a study of tympanic anatomy (Fig. 104), that pus collecting in the tympanic vault must soon find its way into the antrum. The antrum, therefore, is usually to some extent involved in a suppurative inflammation of the vault, and sensitiveness to pressure over the antrum is rarely altogether absent from the most acute stage of an acute purulent otitis media.

Course of the Disease.—The course of an uncomplicated case of acute suppurative otitis media is somewhat similar to that of the acute "catarrhal" form, but is usually more prolonged. Some very severe cases, however, make surprisingly rapid recoveries. The discharge—increasing during the first day or two following myringotomy—is usually very profuse at first, its duration depending upon the patient's constitutional condition, the virulence of the infection, and the extent of the area involved in the vault and antrum. The best possible incision through the drum membrane may not provide perfect drainage of the membranous pockets of the vault, and can not adequately drain the antrum. The process of resolution includes, therefore, the removal of pus in part through an incision or perforation of the drum-head, and in part by its elimination through the lymphatic system. In a favorable case, the discharge may cease and the drum membrane close in a period varying from ten days to four weeks.

Acute Otitis Media in Infants.—Before leaving the discussion of symptoms, a word should be said as to the diagnosis of acute middle-ear inflammation in infants and young children.

It has been frequently stated in otological literature that acute otitis media in young children is invariably announced by a considerable elevation of temperature. This is not strictly true. In the Willard Parker Hospital for Acute Infectious Diseases, the writer has seen a number of cases in which an acutely inflamed ear with bulging of the drum membrane has coexisted with a normal or but slightly elevated temperature. Such cases, however, form so small a percentage of the total number of cases in young children, that they are to be regarded, perhaps, as the exceptions which prove the rule. They are mentioned simply to emphasize the fact that one can not with certainty exclude acute tympanic disease even in young children by the absence of fever. As a general rule, however, it is perfectly correct to say that in the great majority of cases, even the milder grades of acute middle-ear inflammation give rise to a very considerable rise of temperature.

In a child of three years or less, the subjective symptoms of acute otitis media are often so indefinite as to be of little or no diagnostic value to the general practitioner. So skilful and experienced an observer as Dr. C. G. Kerley[6] has stated his belief that young children do not, as a rule, experience earache with acute middle-ear inflammation. While I have never been able to accept this view as quite justifiable, it is certainly remarkable how

[6] Kerley: Symptomatology of Acute Otitis in Children, New York Medical Journal, July 8, 1905.

long a bright little child may suffer from an acute tympanic lesion without directing attention to the ear. If earache is not always an available symptom, it is obvious that such subjective phenomena as tinnitus aurium and moderate impairment of hearing are often quite indeterminable in young children.

If I were called upon to enumerate the symptoms of acute tympanic disease in young children, I should give them in somewhat the following order:

1. *Fever:* high temperature—102° to 105° F.—is present in the great majority of cases. Unexplained temperature should, therefore, call for prompt examination of the ears.

2. *Earache*, when complained of, is, of course, the most useful subjective symptom.

3. *Restlessness*, shown by irritability of temper, peevishness, loss of interest in play, crying without apparent cause, sleeplessness at night, or restlessness (tossing) during sleep—one or any of these symptoms should, unless otherwise accounted for, be regarded as pointing to possible tympanic disease.

4. *Grabbing at the ears*, or rubbing the ears, during sleep or during the waking hours, is regarded as a sign of tympanic pain, and undoubtedly often helps to locate a tympanic lesion. It is often, however, a sign simply of restlessness from other causes, examination showing normal ears. It may, therefore, be a useful sign, but is by no means pathognomonic.

Besides those above mentioned, there are of course an army of symptoms which may be present during acute tympanic disease, but which can not be described as characteristic thereof. I believe very firmly that the routine practice of examining the ears of all sick children would prevent not only much unnecessary suffering, but also some loss of life.

Complications of Acute Purulent Otitis Media.—

1. Acute suppurative mastoiditis: which may give rise to
   (a) Infective sinus thrombosis.
   (b) Epidural abscess.
   (c) Cerebral abscess.
   (d) Cerebellar abscess.
   (e) Acute diffuse suppurative labyrinthitis.
   (f) Acute circumscribed suppurative labyrinthitis.
   (g) Purulent leptomeningitis.
2. Acute serous labyrinthitis.
3. Acute diffuse suppurative labyrinthitis: which may lead to
   (a) Epidural abscess.
   (b) Cerebellar abscess.
   (c) Purulent leptomeningitis.
   (d) Infective sinus thrombosis.
4. Direct infection of the jugular bulb (rare).

The above are given in what I believe to be the order of their frequency. Some of the lesions included under acute mastoiditis might, theoretically,

be included among the sequelæ of acute purulent otitis media. They occur so rarely, however, without intermediate infection of either the mastoid process or the labyrinth, that the above scheme gives a much more correct view.

Acute circumscribed suppurative labyrinthitis is included among the possible sequelæ of acute mastoiditis, but not among those of acute purulent otitis media, because direct infection of the labyrinth from a tympanic lesion usually proceeds through one of the labyrinthine windows, and is almost invariably diffuse. Acute circumscribed suppurative labyrinthitis results most often from necrosis of one of the semicircular canals, the horizontal canal being the commonest point of attack. This practically never results directly from an acute tympanic lesion. Infection of the jugular bulb as a direct result of purulent otitis media—*i.e.*, without intermediate infection of the mastoid cells—is a somewhat rare condition, of which a series of cases have been reported by McKernon.[7]

TREATMENT.—Most cases of acute tympanic disease are amenable in some degree both to *general* and to *local* treatment. We shall speak first, therefore, of such general measures as are of value in the management of any case of acute otitis media, and then of the local treatment appropriate to each of the two main varieties of the disease.

General Treatment.—When the aural lesion occurs as a complication of some systemic disease (*e.g.*, diphtheria, scarlatina, etc.), the general treatment must obviously include the treatment of that disease. When not secondary to an infectious disease, the general treatment resolves itself into an attempt to place the patient under the most favorable conditions for combating the local infection. In uncomplicated cases, this may be summed up practically in the following indications: (a) rest, (b) catharsis, (c) regulation of the diet, and (d) hygienic arrangement of the sleeping room.

Rest.—By this is meant not merely absence from school or business, but absolute rest in bed. This favors recovery in two ways: (1) it secures freedom from the fatigues and excitements of the daily routine, which in the presence of an acute inflammatory lesion entail more than ordinary strain; and (2) it protects the patient from sudden changes in the peripheral body temperature. This is a matter of considerable importance in acute aural disease, where slight accessions of nasopharyngitis may cause rapid extension of the tympanic lesion. With children, it is particularly important and should be insisted upon.

Catharsis.—In hospital practice, the routine treatment begins with the administration of some efficient laxative. Calomel in doses of gr. j to gr. iij according to the patient's age, and followed in six hours by some drug acting more directly upon the intestines, gives the best results. This routine measure should be prescribed regardless of the presence or absence of constipation or digestive disorder.

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[7] McKernon: Primary Jugular Bulb Thrombosis in Children as a Complication of Acute Purulent Otitis Media, N. Y. Med. Jour., July 1 and 8, 1905.

Diet.—The digestive function is apt to be easily disturbed during the acute stage of any suppurative lesion. It is a common practice, therefore, to place a patient suffering with acute tympanic disease upon a strictly liquid diet. This, during the first twenty-four hours, is probably, in severe cases, a wise measure. As soon, however, as the patient begins to demand a more generous diet, it should be changed to a light diet of easily digested solid food, from which red meats should, at first, be excluded. Nothing is more senseless or irrational than to deplete the patient's strength and powers of resistance by a prolonged starvation diet.

Arrangement of the Room.—With most cases of acute tympanic disease there is a coexisting tubal catarrh with more or less pronounced naso-pharyngeal congestion. A room overheated tends rather to increase nasal congestion, and is as bad, if not worse, than one in which the temperature is too low. With the patient in bed, the room should be well ventilated, and the temperature in winter kept at about 60° to 65° F.,—the bed, of course, being so placed as to be protected from draughts. Unless these matters are arranged for by explicit directions, it will be no uncommon experience to find at one's second visit all the windows closed and the temperature of the room at 75° or 80° F.,—a condition in itself capable of inducing pyrexia in one already ill.

Non-operative Treatment of Acute Otitis Media.—By this is meant the management of a case of acute middle-ear inflammation without incision of the drum membrane. It is indicated in only a comparatively small percentage of cases as they are first seen by the otologist.

When inspection of the drum-head shows the lesion to be in the incipient stage, it may be advisable to try to abort the attack without incising the drum membrane. This is sometimes spoken of as the "abortive treatment." It is admissible only in cases in which there are no evidences of pus accumulation or retention within the tympanum,—*i.e.*, in which the drum membrane, though red, is not bulging.

The abortive plan of treatment is as follows: The patient, whether child or adult, is at once ordered to bed. Calomel in dosage appropriate to the age is administered, and followed in six hours by some drug acting directly upon the bowels. This cleansing of the alimentary tract seems not only to prevent or relieve digestive disturbances, but also in some way to exert a favorable influence upon the tympanic lesion itself.

To control pain while the abortive remedies are given time to act, the following prescription is of value, and in itself may influence the lesion favorably:

℞ Codeinæ,
Extract. belladonnæ, āā gr. j;
Phenacetini,
Salol, āā gr. xx.
M.—Divid. in chart. No. viii.
Sig.—One powder q. 4. h.

During the first twenty-four hours the patient should be placed on a strictly liquid diet, which is then changed to a light diet of easily digestible

food. Regular diet should be restored as soon as the lesion seems under control. It is certainly irrational to keep a patient suffering from acute middle-ear inflammation upon a continued starvation diet.

Relief of Pain.—The meagreness of our resources for the relief of pain constitutes the weak point in this plan of treatment. Opium for the relief of the earache of acute otitis media is to be advised against for the reason that it would mask a very important guide as to the progress of the lesion. Locally our chief reliance is upon the application of dry heat, which, while it may not wholly annul a severe earache, often reduces very materially its intensity. While many appliances have been contrived for applying dry heat to the ear, none is more efficient than the ordinary hot-water bag. It should be only half filled with very hot water, wrapped with one or more layers of flannel, and the patient instructed to lie with the diseased ear in contact with it. With two bags in use, continuous application of heat is easily maintained. Poultices applied to the ear are said sometimes to affect the organ disastrously, and are never used in otological practice.

When these measures seem at the end of twelve or twenty-four hours to have controlled the symptoms,—*i.e.*, when the pain is relieved and inspection of the drum membrane reveals no evidences of pus accumulation within the tympanum,—the abortive plan of treatment should be continued. In cases responding favorably to this treatment, the pain usually subsides fairly promptly. The subjective noises, on the other hand, may be very persistent, and restoration of the drum membrane to its normal condition and appearance is often very gradual or slow.

A possible disadvantage in this plan of treatment is illustrated in certain cases in which recovery, though ultimately complete, has been unduly delayed. There have been cases also in which, after a considerable period during which a favorable result seemed probable, incision of the drum membrane has finally become necessary on account of recurrence or persistence of symptoms.

When pain is not relieved within a reasonable period of time, or when the drum membrane, from having been simply injected, is seen to bulge into the canal from the pressure of pus or serum behind it, abortive measures should be abandoned.

That this plan of treatment is not more often put into practice by the otologist is due to two facts,—viz. (1) in most cases the patient does not come under his care and observation until after the lesion has passed the stage for which abortive measures are appropriate, and (2) the pain is often so great that the patient demands or requires immediate relief, and this is most promptly and safely secured by incision of the drum membrane.

Surgical Treatment of Acute Catarrhal Otitis Media.—When the lesion has advanced to the stage of pus formation and retention, it is clearly a surgical condition. The local treatment in such cases is logically based upon a recognition of two facts: (1) the necessity of providing free drainage from the tympanum by means of an incision of the drum membrane, and (2) the necessity of keeping the external auditory canal as nearly as

possible free of pus. We endeavor to carry out the second indication by (a) frequent irrigation of the meatus with sterile or antiseptic solutions, or (b) by capillary traction through gauze wicks or some form of sterile gauze dressing introduced into the meatus.

Myringotomy. — When inspection of the ear shows the membrana tensa to be bulging (Fig. 102, p. 163), there should be no unnecessary delay in making a free incision through its posterior segment. This operation, though occupying but a few moments, is exceedingly painful. It is much better, therefore, that the patient should be under the influence of a general anæsthetic. For this purpose nitrous oxide is the ideal drug, providing safe anæsthesia easily prolonged to the requirements of an operation on one or both ears, and usually leaving the patient absolutely without discomfort or untoward symptoms. The necessary steps in preparing the patient for operation are simple. Having been put to bed, where for the present he is to remain, the auditory canal is filled with peroxide of hydrogen. This is allowed to remain some three or four minutes, the ear then being irrigated with a warm antiseptic solution,—*e.g.*, bichloride of mercury 1 in 4000, or carbolic acid 1 in 200. A bit of sterile absorbent cotton or sterile gauze is introduced lightly into the outer extremity of the canal, and the patient is ready for the anæsthetic.

Since our object is to evacuate pus presumably confined to the atrium, it is not either necessary or desirable that the incision should extend above the posterior fold so as to enter the vault. It is quite important, however, that the cut should be as extensive as possible within the limits of the membrana tensa. The posterior segment of the tense membrane is the broader, and is usually the part most markedly bulging in acute tympanic disease, and it is in the posterior segment that the incision is always made. A mere puncture of the drum membrane or a very short incision may bring temporary cessation of pain through relief of tension, but either closes prematurely or provides such inadequate drainage as not to insure the best results. Another unquestionable disadvantage of a mere puncture of the drum membrane is the fact that, even should it not close prematurely, it is much more apt to result in a permanent perforation than a more extensive incision. The incision should extend from the lower attachment of the posterior segment below to the middle of the posterior fold above (Fig. 109).

Fig. 109.—Incision of membrana tensa.

Removal of Clot following Myringotomy.—Immediately following incision of the drum-head, there is always a free escape of blood into the meatus, and unless this receives attention, a clot may be left in the depths of the canal, partly or wholly defeating the purposes of the operation. I have known this to occur in cases left to the care of incompetent nurses. It is likely to occur, for example, if the first irrigation is delayed twenty minutes or more after the incision is

made, and then the fountain bag instead of the hand syringe is used. I personally like to remain five or ten minutes after operating to see this post-operative clot, which is invariably present, dislodged. Once removed, it does not usually re-form.

From this stage, the treatment is much the same as that described as the abortive method, plus measures for keeping the canal free of pus. The patient should be kept in bed,[8] placed upon liquid or very light diet, and the bowels freely moved.

Irrigation.— During the first few days after myringotomy, the ear should be irrigated at rather frequent intervals with some antiseptic solution. Ordinarily, every three hours is often enough, but if the discharge becomes unusually profuse, syringing every two hours may give better results. Many bactericidal drugs have been advised by different authors, bichloride of mercury and boric acid being in most common use.

Corrosive Sublimate in Aural Therapy.—Admitting that the use of this drug in solutions of proper strength is often followed by satisfactory results, there have in my experience been so many cases which seemed to be unfavorably influenced by it, that I am tempted to record my objections to its routine use. The unfavorable reactions as I have observed them may be mentioned in somewhat the following order: (1) In a certain small percentage of cases corrosive sublimate even in weak solution has seemed to exert a distinctly destructive action upon the tissues, the incision in the drum membrane showing little tendency to heal, but rather to further enlargement by tissue disintegration. (2) There is a class of cases, particularly numerous among children, in which a bichloride solution of any strength produces a dermatitis of the auditory canal and concha, resulting in an eczematous eruption which will persist so long as bichloride irrigations are continued. (3) Finally there have been cases in which the aural discharge has without other discoverable cause been unduly prolonged, improvement following quickly upon a change of treatment.

The use of this drug and the phenomena above described have seemed to me often to occupy a logical relation of cause and effect. Bichloride of mercury in 1 to 5000 solution, applied frequently to the hands, will produce a dermatitis. Taken into the stomach, it would not only prove toxic by absorption, but would quickly produce severe gastro-enteritis by its local action upon the mucosa. Entering the tympanum through the incision in the drum membrane, it is probable that the advantage derived from its bactericidal action is more than offset by its corrosive action upon the tympanic mucosa. Certainly many cases of acute otitis media—particularly in children—do better as soon as bichloride of mercury is discontinued.

Personally, the writer believes that in the cases in which routine irrigation is successfully employed, its happy effects are due very largely to

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[8] While it is not always possible to insist upon confinement to bed in the case of a busy man or woman, there can be no doubt that it may play an important part in hastening recovery, and that its neglect is not without risks to the patient.

its mechanical results in cleansing the meatus of pus, and only in minor degree to the germicidal action of the drug used in solution. He therefore prefers boric acid as less irritating than bichloride of mercury, and is inclined to believe that water absolutely sterilized by boiling might prove as effective as either.

As soon as there is a noticeable diminution in the discharge, the frequency of the irrigations should be reduced,—*e.g.*, three times daily, twice daily, once daily, etc.

Practical Suggestions.—I believe that every practical aurist will agree with me that the value of routine irrigation in acute middle-ear disease depends much less upon the frequency than upon the thoroughness of the irrigation. The object in view is simply the complete removal of pus from the meatus. If this can be accomplished by a single forcible compression of a small rubber syringe, we gain nothing by repetition of this procedure. If the contents of a vessel containing two quarts—*e.g.*, the rubber bag of the ordinary fountain syringe—have been allowed to flow in and out of the ear, leaving a residue of thickened pus at the fundus of the canal, nothing of value to the patient has been accomplished. With an adult, some form of hand syringe by which current force can be controlled gives satisfactory results. The auricle should be drawn somewhat upward and backward, and a stream of some force directed along the posterosuperior canal wall toward the drum membrane. Properly used, the contents of an ordinary coffee-cup usually suffice thoroughly to cleanse the canal of pus. After the irrigation, a ball of sterile absorbent cotton should be placed in the concha (not introduced into the meatus), and the head turned sideways so that the irrigated ear is directed downward. The cotton is removed as soon as the surplus moisture from the irrigation has been absorbed.

With nervous or very young children, the hand syringe is often difficult for the nurse to manage effectively, and then the fountain syringe may be used instead. When the fountain syringe is employed, it is better to use a larger quantity of water,—*i.e.*, one or two quarts,—and the bag should be held or secured on an elevation at least three feet above the patient's head.

I have known a vigorous infant to resist even the fountain syringe so effectively as to make irrigation very difficult even by this method. In such a case the child should be tightly wrapped in a sheet, the hands and arms being pinioned to the side of the body, and the sheet held by safety-pins. By this procedure resistance is rendered impossible, and it is surprising how soon the most unmanageable child realizes the painlessness of the measure and ceases to object to it.

No matter how carefully the nurse may perform her duties, it will be found that the fountain syringe will in certain cases leave a residue of thickened pus at the bottom of the canal. This is a common cause of failure in children. It is due often to the thick, adhesive character of the pus rather than to a faulty method of syringing the ear. This difficulty may be eliminated by preliminary use of a few drops of hydrogen peroxide

poured into the meatus and allowed to remain four or five minutes previous to the irrigation. The value of the peroxide is not so much from its bactericidal action as from its power of dissolving thickened secretions at the fundus of the canal, thus facilitating their expulsion. The theory that this use of peroxide of hydrogen may cause a spread of the infection by the evolution of gas within the tympanum is not supported by any observed facts in actual practice.

When satisfactory results do not follow the irrigation method of cleansing a discharging ear in acute tympanic disease, we should look for a possible cause of failure in one or other of the following conditions, viz.:

1. *Faulty Technic.* — Direct the nurse to irrigate the ears as usual, and immediately thereafter examine the ears by speculum and reflected light. If a residue of pus remains in the depths of the canal, the procedure is worse than useless. This is undoubtedly a common cause of failure.

2. The discharge may be so profuse as to require more frequent irrigations than are being employed. In this case, increasing their frequency to the point necessary to keep the meatus free of pus may give better results.

3. The incision in the drum membrane may have prematurely closed to a point no longer adequately draining the tympanum. While gradual closure of the perforation belongs to the normal process of repair, yet, if it occurs without proportionate diminution in the amount of pus secreted, a second incision of the drum membrane may be necessary.

4. Finally, there are certain cases in which one is forced to conclude that frequent irrigations tend to retard tympanic resolution. Such a conclusion being reached, the irrigations should be discontinued or modified by combination with other measures.

Wick Treatment in Acute Otitis Media. — Personally I believe that there are few cases of acute middle-ear inflammation which do not sooner or later reach a stage in which the wick treatment may with advantage be substituted for irrigation, or combined with it. By this method we depend partially upon capillary traction by means of sterile gauze to take up the pus draining into the meatus.

Technic. — Free drainage from the tympanum being assured, the meatus is cleansed by means of pledgets of sterile absorbent cotton wound about applicators,—first dry and then dipped in alcohol. A prepared wick or strip of absolutely sterile gauze is then carefully introduced into the meatus so that its end is in contact with the perforation in the drumhead. The whole canal is thus filled with gauze, lightly packed. The outer end of the wick or gauze strip should not protrude from the meatus. The concha should now be filled with a small pad of sterile absorbent cotton. In the case of a restless child or nervous adult, who would be likely to disturb this dressing, the wick may be allowed to protrude from the meatus, and this and the whole ear may be covered with handkerchiefs of sterile gauze such as are used in covering a mastoid wound, the whole being held in place by a narrow gauze bandage. With the average intel-

ligent adult, however, the simpler dressing answers every purpose and possesses some advantages. Within twelve to twenty-four hours, this dressing should be removed and the wick carefully examined for the amount of pus that has come away with it. Frequently the wick and cotton in the concha are found completely saturated with pus at the first change of dressings. This, of course, is what one would expect. The meatus is now cleansed as before with alcohol and sterile absorbent cotton, and the drum membrane inspected to make sure that the incision is not prematurely closing. Drainage being adequate and the patient's general condition being satisfactory, a sterile wick exactly similar to the first should be applied. These dressings should be changed at regular intervals of twelve to twenty-four hours, according to the copiousness of the discharge. Each dressing removed should be carefully scrutinized for changes in the amount of pus. In a favorable case, a distinct diminution will soon be noticeable. The use of wicks may be combined with irrigation by syringing the ear at each change of dressing, a plan which often gives exceedingly satisfactory results. In some cases it will be found necessary to abandon the use of wicks altogether. In the writer's hands, however, it has been of undoubted value in shortening many attacks, and he is convinced that it has turned the scale in some cases which were not progressing favorably.

The chief disadvantages in the wick treatment are the difficulties in correctly carrying out its provisions. It can be employed safely only in such cases as can be kept under the physician's close personal care and supervision. The writer regards it as contra-indicated in (a) acute infectious diseases complicated by acute middle-ear inflammation, and (b) in cases of acute tympanic disease accompanied by high fever or other signs of septic absorption.

Catheter Inflation in Acute Tympanic Disease.—The Eustachian catheter has a distinct field of usefulness in the treatment of acute middle-ear inflammation. That it is so seldom used is due probably to the fear that the current of air may carry pus from the tympanum into the mastoid cells. This, in the writer's opinion, is a remote possibility. In the normal ear the atrium is more or less separated from the vault by the structures massed at the boundary line between the two (see Fig. 27). During an acute middle-ear inflammation the swelling of the tympanic mucosa must tend to render this separation more complete. If inflation were performed in a case in which no adequate opening in the drum membrane had been provided, it is possible that harm might result from the displacement of pus. After free incision of the drum-head, however, it seems clear that air entering the tympanum from the Eustachian catheter must find its path of least resistance through the incision in the drum-head, carrying with it some of the pus or fluid in the tympanum. Upon physical grounds, therefore, it would seem that catheter inflation may be used to advantage in certain stages of acute tympanic inflammation. For example, when diminution in the amount of discharge has warranted us in reducing the number of irrigations to twice or three times daily, we have reached a

point from which further progress is in some cases rather slow. Suppose, in such a case, that after irrigation we inspect the drum membrane and, by wiping out residual moisture, satisfy ourselves that the canal is free of pus. We may now cleanse the nose and nasopharynx by alkaline sprays and carefully inflate the ear by catheter, using not too much force. If subsequent inspection shows a residue of pus thrown out into the meatus, we may easily wipe this out with sterilized cotton. If now we introduce a sterile gauze wick into the canal and leave this in contact with the drum-head to take up pus as it escapes through the perforation, it is clear that we have left both canal and tympanum in a condition more favorable for prompt resolution than if irrigation alone were depended upon. I am convinced that the course of the disease may in many cases be materially shortened by these measures.

Fig. 110.—Incision indicated in acute purulent otitis media.

Treatment of Acute Purulent Otitis Media.—The treatment of acute suppurative otitis media may be dealt with briefly, since it is necessary only to point out wherein it differs from that of the milder form. In the first place, there is no stage of the disease for which palliative, or abortive, measures are either adequate or safe. Infection of the vault is in itself an indication for incising the drum membrane. In practically all cases, therefore, the treatment begins with a myringotomy. The incision should begin near the lower marginal attachment of the posterior segment below, and extend upward to and through the posterior fold so that the knife may enter and provide drainage from the vault (Fig. 110). When, in addition to the inflammatory changes in the drum membrane, the posterosuperior canal wall is noticeably inflamed or swollen, this also should be incised. This is accomplished by rotating the knife so that its cutting edge is directed backward, and dividing the adjacent inflamed portion of the canal wall as the blade is withdrawn (Fig. 111). This inflammatory condition of the meatus resulting from acute suppurative otitis media is practically always confined to the soft parts covering the posterosuperior

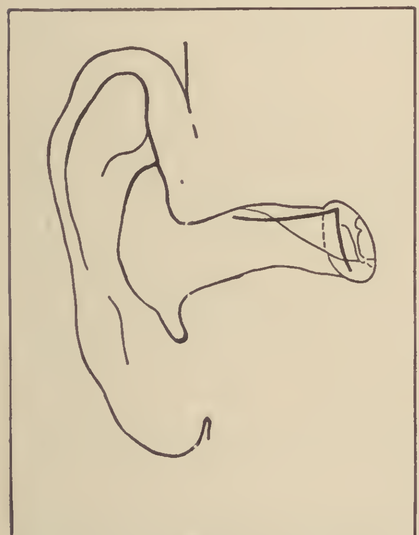

Fig. 111.—Incision through drum-head and posterosuperior canal wall.

wall of the bony meatus. The incision, to give maximum relief, should divide all tissue down to the bone.

Following myringotomy, the measures advocated for the milder form of the disease are of equal or even greater importance here. Absolute rest in bed, catharsis, and careful regulation of the diet may exert a decided influence upon the course of the disease, or at least upon the duration of the attack. The ear should be irrigated every three hours with a sterile or antiseptic solution,—a boric acid solution (dr. ss ad oz. viij) being the writer's preference. Later, when the amount of the discharge has become noticeably diminished, the frequency of the irrigation should be reduced,—*e.g.*, to one, two, or three times a day. I believe that the ear should not be syringed oftener than is necessary to keep the canal fairly free of fluid pus. With diminution in the amount of the discharge, the use of gauze wicks may be combined with periodic irrigation. By this plan the ear is irrigated once or twice daily,—once being usually sufficient,—the canal in the interim being filled with a strip of sterile gauze. In addition to the above measures, occasional catheter inflation to clear the atrium and hypotympanic space will influence some cases favorably. It must be employed with care,—*i.e.*, with only moderate force,—and only after the acute stage is well passed, the physician having assured himself that an adequate opening in the drum membrane remains. The objection that inflation, even though practised with gentleness, may in these cases carry pus from the antrum to the mastoid cells is, I believe, founded on theory rather than fact.

Antrum Tenderness.—At the height of an acute suppurative otitis media of average severity, some degree of tenderness over the antrum is usually present. This physical sign undoubtedly denotes an extension of the inflammatory process from the tympanic vault backward to the antrum, but does not necessarily mean suppurative involvement of the mastoid cells proper. I believe that this condition—*i.e.*, tenderness localized over the antrum and occurring early in a suppurative middle-ear lesion—is one for which the application to the mastoid of the ice-bag or Leiter's coil may be of positive value in limiting the process and preventing spread of infection throughout the mastoid cells. All, however, that can be accomplished by its use is obtained by twenty-four hours of continuous application, after which it may be distinctly harmful in lowering the vitality of the part.

Occasional Necessity for Re-incising the Drum Membrane.—The normal drum membrane exhibits a remarkable tendency to rapid healing after puncture or incision, provided infection has not occurred. I have seen a drum membrane, after free incision for the relief of severe earache in the initial stage of acute catarrhal otitis media, practically healed at the end of forty-eight hours. This tendency to quick healing occasionally works to our disadvantage in the treatment of acute purulent otitis media, in which closure of the incision advances more rapidly than repair of the suppurative process within the tympanum. I wish to emphasize the fact that

in cases of retarded resolution following myringotomy, if inspection shows that the opening in the drum-head is no longer affording adequate drainage, there should be neither hesitation nor delay in re-incising the membrane. Nor can we formulate any rule as to the number of times it may be necessary or wise to repeat this operation. Even in cases in which myringotomy has been repeated one or more times, should evidences of pus retention within the tympanum recur, reopening of the drum membrane will often lead to recovery. There can be no doubt that delayed resolution is due in a certain proportion of cases to the fact that the individual's vitality or power of resistance is below par, and it is a reasonable hypothesis that the same causes might unfavorably influence the process of repair following operation upon the mastoid.

The measures outlined above, if thoroughly and conscientiously carried out, will bring about recovery in all cases of acute middle-ear inflammation except in those in which the process of repair is interrupted by one or other of the following conditions:

(a) Acute suppurative mastoiditis.

(b) Caries of tympanic structures leading to the condition known as chronic suppurative otitis media.

(c) Spread of the infection to the labyrinth or to one or other of the intracranial structures. The symptoms by which such extension of the disease is announced will be discussed in connection with the lesions mentioned.

After Treatment.—After the discharge has ceased and the drum membrane is completely healed, the after treatment, if any be required, is the same whether the lesion has been of the catarrhal or purulent type. If the hearing is perfect and no subjective symptoms persist, no treatment is called for. On the other hand, if tinnitus or other subjective symptoms persist, or if the hearing remains below par, it may be necessary to practise occasional inflation by catheter to break up newly formed adhesions and correct any remaining congestion within the tympanum and Eustachian tubes. Quite often, however, these conditions correct themselves without treatment.

ACUTE MASTOIDITIS.

Etiology.—Acute suppurative mastoiditis is practically always secondary to a suppurative lesion of the middle ear. While cases have been reported in which the drum membrane has presented no signs of inflammation, past or present, there is no evidence in such cases that a tympanic lesion has not preceded, and given rise to, the mastoid disease. Primary mastoiditis due to syphilitic or tubercular infection, while theoretically conceivable, is so rare as to be practically negligible. It is not seen in actual practice. Traumatic mastoiditis is among the rarest lesions. In short, the mastoid process seems to enjoy immunity from acute disease except as a result of infection from a diseased tympanum.

Pathology.—We shall obtain a much clearer conception of the pa-

thology of this rather grave lesion if we bear in mind two facts, namely: (1) The so-called mastoid antrum should not be regarded as one of the mastoid cells, but simply as the posterior end of the tympanic vault. In the fœtus at term, the mastoid process as such does not exist, yet the antrum is already a large and easily demonstrable cavity. (2) In acute purulent otitis media, the inflammatory process and the flow of pus must quickly invade the antrum. This, however, does not of itself constitute a true suppurative mastoiditis. Acute mastoiditis may be defined as an acute inflammatory process originating in the antrotympanic cavity and spreading thence to the mastoid cells proper. It is probable that the inflammatory process passes in most cases through certain fairly well defined stages,—viz. (1) stage of vascular engorgement and cell infiltration; (2) suppurative stage, characterized by the presence of fluid pus; and (3) the stage of osseous necrosis or softening. These three stages are clearly and easily recognized macroscopically during operations upon patients in different stages of the disease.

Probably there is no well recognized lesion which presents such wide variations in the rapidity or slowness with which the disease advances. Thus, in one case weeks may elapse without the development of symptoms justifying a decision to operate, while in another case, operated upon but a few days after the onset of the tympanic disease, the cells throughout the mastoid may be bathed in pus. If one will refer to Plate III, page 33, showing recognized structural variations, or types, of the normal mastoid,—or, better still, if one will examine any fairly large series of bone sections,—these variations in the pathology of the disease will be more easily understood. Thus, it is clear that in a temporal bone characterized by a large antrum in close relation to large pneumatic spaces separated only by thin lamellæ of bone, a suppurative process would logically advance with far greater rapidity than in a bone presenting a very small antrum and a sclerotic mastoid.

On exposing the mastoid cortex, we find in a majority of cases no changes indicative of the pathologic changes within the bone. In adults, only if the lesion is of comparatively long duration, are cortical perforations likely to be present. The most common site of such cortical defects is the space slightly above and behind the spine of Henle; but they may occur at any point upon the outer cortex from the temporal ridge above to the tip below. Perforation may also occur through the thin bony plate forming the inner covering of the mastoid tip (Bezold's perforation or abscess).

In young children, owing to the comparative thinness and softness of the outer covering, cortical perforations are by no means uncommon.

On removing the cortex, we may find any of the following conditions, which, in the order named, represent roughly the successive stages of the lesion: (a) Antrum more or less filled with fluid pus draining backward from the tympanic vault. The mastoid cells contain no pus, but their mucous lining is red, swollen, and bleeds easily and pro-

fusely, showing great vascular engorgement. (b) Cells adjacent to the antrum, and also the large cell at the tip, contain a variable amount of fluid pus. (c) Mastoid cells throughout contain pus and exuberant granulations. (d) Intercellular bone substance is softened or completely disintegrated. (e) In the later stages necrosis of the intercellular bone substance may co-exist with intact inner plates, or the latter may exhibit areas of necrosis.

Pus, being present within the mastoid, may be removed wholly or in part by absorption through the lymphatics, or may find a pathway of escape through perforation of the bone brought about through osseous necrosis. The various pathways of escape, and the lesions to which they give rise, will be spoken of in a later chapter.

Symptoms.—The symptoms of acute mastoiditis are usually engrafted upon those characteristic of acute purulent otitis media. We have, therefore, to ask ourselves what symptoms, or modifications of symptoms, occurring during an attack of acute middle-ear suppuration, or during convalescence therefrom, should lead us to suspect suppurative involvement of the mastoid cells.

Pain and Insomnia.—The patient, who has been comparatively comfortable, may suddenly experience deep-seated pain in the region of the mastoid process. Or, in the absence of actual pain, there may be a distressing sense of fulness, soreness, or discomfort referred to the mastoid. On the other hand, there are many cases in which the patient complains little either of mastoid pain or discomfort.

In the presence of mastoid pain or discomfort, and in proportion to its severity, insomnia is usually present, and sudden inability to sleep at night is in some cases a useful guide to arrested resolution.

Fever.—Elevation of temperature, usually of moderate degree, may be more or less continuously present during suppurative disease of the mastoid. When present it is a most important symptom, and one which, unless yielding fairly rapidly to rational non-operative measures, calls more or less imperatively for surgical intervention. Fever, however, is in one sense a most unreliable symptom, in that many cases of severe mastoiditis exhibit throughout no noticeable elevation of temperature. That extensive necrosis of the mastoid cells may coexist with a practically normal temperature curve is a fact now fortunately recognized. In the author's experience fever is absent, or so slight as to be of no special diagnostic value, in a majority of cases. Absence of fever is, therefore, no evidence that the mastoid cells are not extensively diseased.

Aural Discharge.—The aural discharge may undergo certain quantitative changes which would lead an experienced aurist to suspect mastoid involvement. For example, a rather copious discharge may suddenly cease, this abrupt cessation being accompanied by no amelioration of the patient's condition and being followed within a few days or hours by a renewed flow of pus. Or, again, a moderate discharge may suddenly or

gradually increase in volume until it becomes necessary upon purely physical grounds to assume involvement of the mastoid cells in order to explain the amount excreted in the twenty-four hours. In still another group of cases, the long persistence of the discharge may of itself force the inference of a focus of disease beyond the limits of the small tympanic cavity.

Changes in the Drum Membrane and Sagging of the Posterosuperior Canal Wall.—While cases have been observed in which the tympanic condition has improved simultaneously with the advance of a suppurative process within the mastoid, such cases are exceedingly rare. As a rule, the drum membrane presents the picture of severe suppurative otitis media. It is usually perforated, red, and markedly bulging in the upper posterior quadrant. That these tympanic changes are often a result of the disease within the mastoid is frequently demonstrated by the rapidity with which the diseased drum membrane regains its normal condition and appearance after operation upon the mastoid. A very common feature of these cases is a noticeable inflammatory thickening of that portion of the posterosuperior canal wall which immediately adjoins the drum membrane, so that the one gradually merges into the other, there being no distinguishable line of demarcation between the two. This is undoubtedly due to a periostitis in this situation, and is so frequently seen in cases of acute purulent otitis media complicated by mastoid suppuration that it is by some authors regarded as pathognomonic of the latter condition. Taken in connection with other symptoms, it is of considerable diagnostic value; alone, it is not a reliable sign of mastoid disease.

Mastoid Tenderness.—Undoubtedly the most reliable sign of acute mastoid disease is sensitiveness to pressure over the mastoid cortex. This varies in different cases from moderate tenderness elicited only by firm pressure to extreme sensitiveness even to very light pressure. In some degree it is usually present in every case of suppurative mastoiditis. In many cases it seems to reach its height during the early (vascular) stage, becoming less marked as these initial inflammatory changes are past. Most aural surgeons have been occasionally surprised in operating upon cases in which intense mastoid tenderness has been present to find an absence of pus, the most conspicuous macroscopic change being extreme vascular engorgement. During the later progress of the lesion, exacerbations of mastoid tenderness may occur, being due probably to an occasional extension of the inflammatory process to cells not hitherto involved, or to the influence of pus temporarily retained under pressure in certain cells.

Undoubtedly, the degree of mastoid tenderness is in some degree related to the type of bone involved. For example, it seems a logical deduction that a suppurative inflammation involving a mastoid process of thin cortex and pneumatic structure (see Plate III, page 33) would give rise to greater sensitiveness to pressure than would a similar process

within a sclerotic mastoid, or one having a very thick cortex. From this relation, one may in some cases reach a practical inference from one's clinical data as to the type of the diseased mastoid, and therefore as to the character of the lesion. Thus, in a case in which symptoms of mastoiditis with very marked mastoid tenderness follow rapidly upon the onset of the tympanic lesion, one may infer with considerable confidence that one has to deal with a pneumatic mastoid having a comparatively thin cortex. On the other hand, with symptoms of more gradual development and with well-marked mastoid pain or discomfort coexisting with very slight sensitiveness to pressure, one would expect to find a mastoid of sclerotic type, or one having a thick cortex. Clearly the latter may be the more serious condition.

It will be seen from what has been written that the clinical picture includes only a limited number of symptoms which can be said strictly to characterize the disease.

To epitomize: The symptoms to be looked for are *mastoid pain*, with consequent insomnia; elevation of temperature; *certain quantitative changes in the character of the discharge; mastoid tenderness*, and bulging of the posterosuperior canal wall. The above rather meagre clinical picture may be said to comprise practically all the symptoms which are characteristic of an uncomplicated case of acute mastoiditis.

Unfortunately, many cases of acute mastoiditis run their course with complete absence of one or more of the above symptoms. For example, neither elevation of temperature nor mastoid pain may be present. When fever is absent and pain inconsiderable, the subjective and constitutional phenomena are naturally not very characteristic. Even in such cases, however, there is usually some feature in the clinical picture which will cause the experienced observer to suspect that recovery is being interrupted by the advance of a suppurative process within the mastoid. Fortunately, there is one physical sign which is seldom or never wholly absent. Some difference in the sensitiveness of the two mastoids to pressure can usually be demonstrated by careful palpation.

There remain to be described certain changes in the position of the auricle and in the contour of the soft parts about the ear, which are among the more unusual accompaniments of mastoid inflammation. Auricular displacement is a comparatively rare result of mastoid inflammation in adults. When present it constitutes a very characteristic deformity. It may be produced in one of two ways,—viz.:

(1) *Subperiosteal Abscess.*—The suppurative process within the mastoid gives rise to a perforation in the mastoid cortex. Pus escaping through this perforation elevates the periosteum from the surrounding bone, and, being confined beneath the periosteum, constitutes what is known as a subperiosteal abscess. As this abscess is usually in close proximity to the posterior attachment of the pinna, the postauricular sulcus is often obliterated and the auricle markedly displaced. If the perforation is behind the centre of the postauricular attachment, the auricle is pushed

directly forward, standing prominently outward from the side of the head. When the perforation is at a higher level, the auricle is displaced both forward and downward (Fig. 112).

(2) *Postauricular Œdema.*—In another class of cases the acute inflammatory process extends through the mastoid cortex without causing a perforation. This extension of inflammation involves the periosteum and overlying soft tissues, which become markedly swollen or œdematous. This condition gives rise to a deformity similar to that above described, though usually less pronounced.

While auricular displacement is in adults comparatively rare as a result of mastoiditis, it occurs very frequently in the mastoiditis of infants and young children. It will also be recalled that a similar deformity is a very common feature of furunculosis of the meatus in adults. The two lesions are very different, however, in their surgical significance, for whereas the postauricular œdema and consequent displacement resulting from furunculosis of the canal are usually relieved by incision of the focus or foci of infection within the meatus, the subperiosteal abscess or œdema resulting from mastoid suppuration is a positive indication for opening the mastoid. Hence the importance of being able to make a correct diagnosis. It may be well therefore to repeat briefly the chief differential points between the two conditions.

Auricular Displacement in Furunculosis.	*Auricular Displacement in Suppurative Mastoiditis.*
1. Common in adults; rare in children.	1. Very common in children; comparatively rare in adults.
2. Pain always severe; increased by movements of the jaw.	2. Pain not usually severe, and may be absent; not influenced by movements of the jaw.
3. Any manipulation of the auricle causes excruciating pain.	3. Manipulation of the auricle causes absolutely no pain.
4. Speculum examination shows furuncular swelling situated always in fibrocartilaginous portion of canal.	4. Inspection of meatus shows absence of inflammation in fibrocartilaginous part of canal. Inflammation, if present, confined to lining membrane of posterosuperior wall of bony canal.
5. Drum membrane may be, and usually is, intact.	5. Drum membrane almost invariably perforated.
6. Discharge, if present, comes from furuncular perforation in membrano-cartilaginous canal.	6. Discharge from the tympanum through perforation in drum membrane.
7. Pressure upon the mastoid, so directed as not to move or disturb the auricle, causes no pain. Pressure at same point, but directed slightly forward so as to disturb the auricle, causes great pain.	7. Pressure upon the mastoid elicits deep bone tenderness.

Bezold's Abscess.—A more unusual complication of suppurative mastoiditis was first described by the late Professor Bezold, and is known as a Bezold abscess. This condition is caused by a perforation in the bony plate forming the inner surface of the tip of the mastoid. It occurs presumably in cases in which the tip cells are especially large and in which

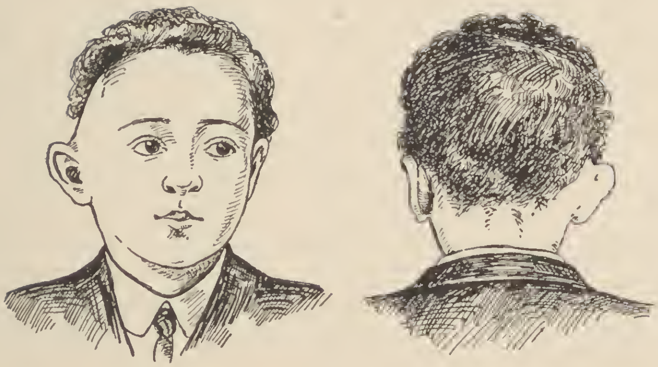

Fig. 112.—Auricular displacement resulting from postauricular subperiosteal abscess.

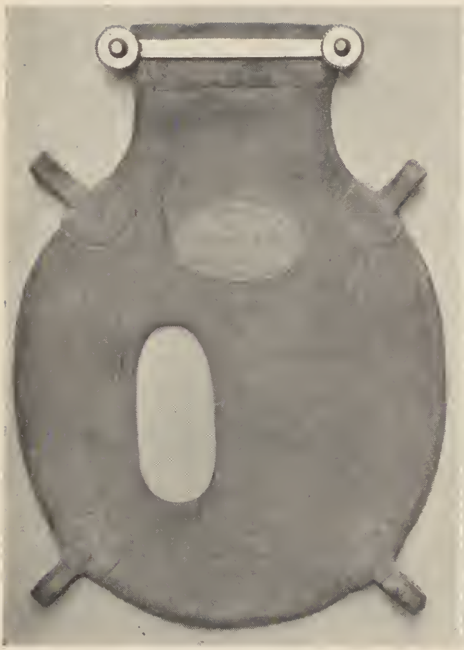

FIG. 113.—Aural ice-bag.

FIG. 114.—Leiter's coil.

the bony plate forming the inner or medial wall of the tip is very thin, and the outer cortex thick. Pus escaping through such a perforation burrows downward in the neck beneath the sternomastoid, or may be confined between layers of the deep cervical fascia. It presents a prominent, elongated swelling below the mastoid in the neck, which, if other symptoms of mastoid disease were not clear, might present difficulties of diagnosis. It renders the surgical treatment of the disease rather more troublesome by the occasional necessity of extensive incisions in order to provide adequate drainage from the cervical spaces involved.

Paralysis of the Abducens (6th) Nerve.—A somewhat puzzling complication, which the aurist has sometimes to deal with, is the development during the course of a suppurative mastoiditis of paralysis of the 6th cranial nerve. It will be recalled that this is purely a motor nerve and that it supplies only one muscle, the external rectus muscle (occuli).

Origin and Position.—According to Gray, the deep origin of the 6th nerve is from the gray substance of the fasciculus teres by a nucleus common to it and to a part of the 7th (facial) nerve. It is said also to be connected with the nuclei of the 3rd and 4th nerves. Its superficial, or apparent, origin is from the upper constricted portion of the medulla just behind the pons. The trunk, as it passes forward along the inner wall of the cavernous sinus to enter the orbit through the foramen lacerum anterius is in more or less close relation with the molar occuli (3rd), the pathetic (4th) and the ophthalmic branch of the trifacial (5th). It is also in relation with the cavernous portion of the internal carotid artery. Within the orbit it is distributed solely to the inner surface of the external rectus muscle, contraction of which rotates the eye outward.

Pressure by tumors, the exudates of a basilar meningitis or of cerebrospinal syphilis may disturb its function.

The diagnosis of 6th paralysis is simple. The patient usually complains of the discomforts incident to double vision. When any small object, *e.g.*, a pencil, is held directly in front of him,—*i.e.*, in the median plane of vision,—it is seen as a single object. So also when it is moved laterally in the direction corresponding to the sound nerve. When, however, it is moved in the opposite direction, diplopia is complained of as soon as the median plane of vision is passed, when it will be seen that the eye corresponding to the paralyzed nerve remains stationary in the median position, while the sound eye follows the moving pencil as normally.

The path of infection from mastoid to 6th nerve is usually obscure. Theoretically, one may say (1) that the nerve may be compressed by disease (thrombosis) of the cavernous sinus; (2) that it may be subjected to pressure by a thrombus in the anterior end of the inferior petrosal sinus (Page's case[9]); (3) that the nerve sheath may be involved in a meningitis spreading inward from a necrotic tagmen antri; (4) that a suppurative labyrinthitis may give rise to a subtentorial leptomeningitis which, extending forward, may involve the nerve sheath; (5) that the nerve lesion may be

[9] Page: Thrombosis of Jugular Bulb; Paralysis of External Rectus Occuli; Annals of Otol. Rhinol. and Laryng., June, 1910.

an extention by continuity from disease involving the apex of the petrous bone (Gradenigo). While some of these roadways of infection seem to have been definitely established in recorded cases, they are usually in actual practice merely hypotheses, which do not throw much light upon the pathologic aspect of the case in hand. In 1918 the author [10] published the history of a case in which a paralysis of the 6th nerve which had existed at least two weeks, disappeared completely on the third day after a mastoid operation; but the pathway of infection could not be determined. The number of such cases which have been recorded is quite large.

In 1910 Dr. C. E. Perkins [11] of New York published a very interesting review of the literature of this subject up to that time. His report embraced sixty cases, in twenty-nine of which, the mastoid was operated upon, no operation having been performed in the remaining thirty-one cases. Of the cases operated upon, there were one death, one failure (*i.e.*, of restoration of nerve function) and twenty-seven complete recoveries. Of the thirty-one cases treated non-surgically there were four deaths, three failures and twenty-five (sic) recoveries. So far as any deductions are permissible from these figures, they would seem to be; that in suppurative mastoiditis associated with 6th nerve paralysis, the mortality is far greater and the percentage of 6th nerve recoveries far less in cases not operated upon than in those in which a mastoid operation has been performed. The author believes, therefore, that even in cases in which the symptoms referable to the mastoid are not very marked, the association of a homolateral paralysis of the external rectus muscle should be regarded as a positive indication for the mastoid operation.

Gradenigo's Symptom-Complex.—Before leaving the subject of abducens paralysis a word should be said as to the surgical import of what is known as Gradenigo's symptom complex. Gradenigo attributed to it a surgical significance only in the presence of a suppurative lesion of the mastoid.

This complex is present when with evidences of middle ear or mastoid infection, there is associated a homolateral paralysis of the 6th nerve, eye pain or facial neuralgia and also severe frontal headache. This is thought to point strongly to necrosis or at least to a suppurative lesion of the apex of the petrous bone.

It is clear that such a diagnosis would place upon the surgeon a very grave responsibility. It is important to bear in mind, therefore, that this syndrome might easily represent the combined results, or symptoms, of separate and distinct lesions. For example, should mastoiditis co-exist, as is not very uncommon, with suppurative disease of the ethmoid cells or sphenoid sinus, it is quite conceivable that the abducens paralysis might be a product of the mastoid lesion, while the sensory components (eye pain, facial neuralgia, frontal headache) might have their origin solely in the diseased ethmoid or sphenoid.

Mastoiditis in Infants and Young Children.—Before leaving the sub-

[10] Kerrison: A case of abducens paralysis complicating mastoiditis: brief discussion of this complication as a surgical indication; Medical Record, November 30, 1918.

[11] Perkins: Abducens Paralysis and Otitis Media Purulenta; Annals of Otology, September 1910

ject a word should be said as to the symptoms as observed in young children.

Since the mastoid process as such does not exist during the first months of infantile life, it seems paradoxical to speak of mastoidectomy as occasionally called for at this period of development. It is an established clinical fact, however, that even in the first months of life, acute middle-ear inflammation may give rise to necrosis of the outer wall of the antrum and consequent subperiosteal abscess, and this condition for convenience of description is spoken of as mastoiditis. Antritis would be a scientifically more correct term.

The symptoms of mastoiditis in infancy and early childhood resemble those of adult life, with the following differences: 1. The child suffering from acute aural disease is usually unable to differentiate between pain referred to the antrum and pain localized in the tympanum. Antrum pain is therefore of little diagnostic value in determining mastoid inflammation.

2. Fever is usually present rather persistently during the acute stage of an acute purulent otitis media. After its recession a subsequent rise of temperature may easily be due to a slight exacerbation of the tympanic lesion. Fever, therefore, is hardly a reliable sign of mastoiditis in children.

3. Occasional symptoms suggesting cerebral irritation,—*e.g.*, convulsions, sudden vomiting, chills, hyperpyrexia, etc.,—while naturally causing anxiety, are of less serious import than if occurring in an adult patient suffering from mastoid inflammation, for the reason that the cerebral centres presiding over these disturbances of function are more subject to reflex irritation from slight peripheral causes than in the adult. Such symptoms are not, however, even in very young children, to be considered lightly, and the child exhibiting them should be very carefully watched.

A conspicuous difference in the Physical signs of mastoiditis in early life depends upon the fact that the outer wall of the antrum in infants, and the outer mastoid cortex in children of two or three years, are softer, more vascular and spongy, and infinitely thinner than in later life. As a result of these structural peculiarities, perforation of the cortex with consequent subperiosteal abscess is a comparatively frequent and early manifestation of mastoid disease in early life. As a matter of fact, the diagnosis of mastoiditis in young children is frequently not made until after the appearance of postauricular œdema or subperiosteal abscess.

Prognosis.—Most cases of acute mastoiditis which are brought under the care of a competent aurist recover without operation upon the mastoid. Of the minority which do not respond to non-operative measures, but which present no evidences of intracranial complications, the prognosis as to recovery after operation is very favorable. Taking any large series of such cases operated upon by competent aural surgeons, the percentage of mortality probably does not exceed 1 or 2 per cent. In those cases in which, either before or after operation, the intracranial structures become involved (brain abscess, meningitis, sinus thrombosis), the mortality is exceedingly large,—depending in part upon the character of the lesion, and in part upon the amount of surgical skill and judgment which are brought to their care.

Treatment.—The patient should be ordered to bed, and kept there until all physical signs of mastoid inflammation have subsided. An active cathartic should be administered. During the first day or two, he should be given a liquid or very light diet.

Early Myringotomy.—Irrespective of a previous incision of the drum membrane, it should again be incised under nitrous oxide anæsthesia, unless tympanic drainage is obviously adequate. The ear should be irrigated with boric acid solution at regular intervals, the frequency of the irrigations being regulated in accordance with the amount of the discharge. A ball of sterile absorbent cotton may, however, be placed lightly in the concha to protect the ear from fresh infection from without, this being changed as often as it becomes saturated with pus.

Application of Cold to the Mastoid.—If the surgeon believes the inflammatory process to be in the incipient stage, the application of an ice-bag or of Leiter's coil may be of positive value in retarding, and perhaps aborting, the attack. It should be used not longer than twenty-four hours and once removed should not be reapplied. After the stage of fluid pus formation is reached, it may reduce the vitality of the part of the degree actually favoring bone necrosis.

The application of leeches to the mastoid, scarification, and blood-letting by means of the so-called "artificial leech," and the old practice of blistering the mastoid, while mentioned in some text-books, are practically without value, and are to be condemned as likely to produce a local sensitiveness to pressure which might obscure an important physical sign,—*i.e.*, either increase or diminution of true bone tenderness.

The administration of opium in acute mastoiditis is particularly inadvisable, for it may not only mask important symptoms, but, if repeated, soon reduces the patient to a condition of nervous irritability unfitting him for anæsthesia and surgical intervention, should these later become necessary.

The successful non-operative treatment of acute mastoiditis depends largely upon the maintenance of free drainage from the vault until the processes of repair within the mastoid are established. If, therefore, during this critical period evidences of premature closure of the postero-superior quandrant of the drum membrane arise, there should be no hesitation or loss of time in re-incising it. The writer has seen many cases in which this simpler surgical measure has saved the patient from the major operation upon the mastoid.

As to the criteria by which the results of non-operative treatment are to be judged, it is best to avoid dogmatism. The patient should, of course, be seen and examined carefully each day. If at the end of three or four days the discharge is less profuse and mastoid tenderness less marked, I should regard these conditions—fever, of course, being absent—as distinctly favorable, and should these conditions show further improvement at the end of a week, I should look upon recovery without operation as probable. On the other hand, if a week passed without improvement in the patient's condition, and particularly if mastoid tenderness remained

unchanged or had become more pronounced, I should feel that the patient's safety called for operative intervention.

The indications for operating in acute mastoiditis may be summed up somewhat as follows:

1. The development during acute mastoid disease of auricular displacement as a result either of postauricular œdema or subperiosteal abscess,—furunculosis of the canal being eliminated as a possible cause.

2. The development during acute purulent otitis media of symptoms of vestibular irritation—*e.g.*, vestibular nystagmus, vertigo, etc.—calls for prompt opening of the mastoid in order to relieve the vestibular walls of the pressure or presence of confined pus.

3. Marked tenderness on pressure, extending well beyond the limits of the antrum, and showing no tendency to diminution within five or six days following the incision of the drum membrane, would point to an active inflammatory process within the mastoid cells, calling for surgical intervention.

4. Marked variation in the quantity of pus discharged, its maximum flow being apparently too great to be explained by the tympanic lesion, its periods of diminution being coincident with the development of mastoid pain or tenderness (or both). Such a combination of symptoms constitutes one of the most positive indications for opening the mastoid.

5. Mastoid symptoms having been present and having disappeared, a discharge from the tympanic vault which resists all rational non-operative measures may, by reason of its persistence, justify the hypothesis of a necrotic area beyond the limits of the antrotympanic cavity. In such cases an operation is often the only means of saving the integrity of the organ and preventing serious impairment of function.

6. Finally, evidences of mastoid disease having been present, the development at any time during convalescence of symptoms of septic absorption—*e.g.*, chills, sweats, septic temperature, etc.—would, in the absence of other concurrent disease, constitute a positive indication for immediate operation.

In speaking of the above as positive indications for opening the mastoid, it must be understood that we are speaking from the view-point of the patient's safety,—*i.e.*, with the view of correcting a lesion which has become dangerous before danger signals proclaim that his life is already in jeopardy. Every aurist has seen cases of mastoid suppuration of the severest type, in which operation seemed clearly indicated, recover without operation. He has also, however, seen patients by whom operation was delayed or rejected, return later with symptoms of intracranial infection. The aural surgeon is, therefore, frequently confronted with a grave responsibility. If he waits in all cases until symptoms are present rendering surgical intervention absolutely obligatory, he will frequently find, when these symptoms appear, that the question is shifted from the advisability of opening the mastoid to a far more serious one,—*i.e.*, the possibility of saving the patient's life by any means at his command.

CHAPTER VIII.

CHRONIC MIDDLE-EAR SUPPURATION.

Otitis Media Purulenta Chronica.—This term is applied to any tympanic inflammation giving rise to a perforation of the drum-head which shows no tendency to heal and through which there is a more or less constant flow of pus.

Etiology.—Probably every case of chronic purulent otitis media has its origin in one or more attacks of acute tympanic disease. As a rule, however, these patients apply for relief only after months or years of constant or intermittent otorrhœa, so that the early changes marking the conversion of the acute into the chronic form of the disease rarely come under actual observation.

All observers who have had an opportunity of watching the ear changes in any large series of cases of the acute exanthemata must have been impressed with the remarkable rapidity with which the drum membrane is frequently destroyed in acute tympanic lesions complicating scarlet fever and diphtheria. In some cases this tendency is so pronounced that nothing the physician may do seems to exert the slightest influence upon the rapid and permanent destruction of tissue. Measles and influenza also contribute their quota to the number of cases of acute tympanic disease which ultimately become chronic. Probably from 18 to 20 per cent. of all cases of chronic middle-ear suppuration are traceable to the infectious diseases of childhood.

Adenoids predispose the child so markedly to acute middle-ear disease and interfere so effectively with spontaneous recovery that they must be included among the active causes of chronic purulent otitis media. This explains in some degree the greater frequency with which the disease develops in childhood as compared with adult life.

An occasional cause of the disease in adult life is found in the atrophic changes in the drum membrane occurring in some cases of non-suppurative middle-ear catarrh. The drum membrane in such cases may be exceedingly thin, transparent, and retracted, or may be apparently thickened and relaxed. Probably most aurists have seen cases in which, during an attack of acute purulent otitis media of apparently moderate severity, the membrana tensa has undergone rapid disintegration about the site of the perforation, which has subsequently shown little tendency to regeneration. The rapid loss of tissue in these cases is probably due to atrophy of the normal fibrous layers of the membrana tensa, which are ordinarily so strongly resistant to the influence of an acute suppurative process within the tympanum.

Giving due weight and consideration to other causes, there can be little doubt that the great majority of cases are directly traceable to neglect or unskilful management of an antecedent attack of acute purulent otitis media.

Pathology.—The drum membrane is necessarily perforated in every

case of chronic purulent otitis media. In the great majority of cases the perforation is central,—*i.e.*, does not at any point involve the marginal attachment to the annulus tympanicus. In size it may vary from a minute pin-point orifice to practical destruction of the greater portion of the membrana tensa. It may also vary in shape from a small circular or oval opening to the larger crescentic or horseshoe-shaped defects involving both anterior and posterior segments. When the membrane bordering the perforation is relaxed and indrawn, and the tympanic mucosa is swollen and denuded of its surface layer of epithelium, adhesions may form between the margins of the perforation and the inner tympanic wall.

Perforation of Shrapnell's membrane seldom or never occurs during acute purulent otitis media, but is found in about 3 per cent. of cases of chronic middle-ear suppuration (Bezold). While an artificial opening in Shrapnell's membrane would lead into Prussak's space, that occurring in chronic purulent otitis media invariably forms a direct communication with the vault, and therefore with the aditus and antrum.

Marginal perforations—meaning thereby perforations in which there is at some point absolute destruction of the peripheral edge—are found in a certain proportion of cases of long-standing middle-ear suppuration. They also occur primarily in certain cases of very rapid destruction complicating profound systemic infection, notably in cases of scarlet fever or diphtheria of unusual severity. Generally speaking, a marginal perforation ot the drum membrane must be regarded as pointing to a more serious lesion of the tympanum than a central perforation, for the following reasons: (1) in a large percentage of cases, a marginal perforation indicates necrotic changes in the bone adjacent to the perforation: (2) the great majority of marginal perforations involve either Shrapnell's membrane or the upper posterior margin of the membrana tensa, in either case leading directly into the vault, aditus, and antrum; and (3) a marginal perforation involving Shrapnell's membrane or the posterosuperior segment of the membrana tensa, or both, constitutes the ideal condition for extension of the epidermis of the meatus into the antrotympanic cavity. This leads in some cases to a proliferation of epithelial elements within the antrum, and provides the most favorable nucleus for the subsequent formation of cholesteatomatous masses within that cavity. According to the author's experience, marginal perforations always mean osseous necrosis, the structures most commonly involved being the malleus and incus and the contiguous portions of the tympanic ring and Rivinian segment.

The Mucosa.—The changes in the mucous membrane may vary in different cases from simple hyperæmia with infiltration of leucocytes to practical destruction. In the simplest form of the disease, the mucosa may be greatly swollen so as to encroach considerably upon the cavity of the atrium, yet may preserve intact its surface layer of ciliated epithelium. This condition may persist for long periods without actual destruction of tissue. In the severer forms the epithelial covering is lost over considerable areas, in which situations granulations form and there is a marked tendency to adhesions with adjacent structures.

Bone Changes.—It is quite possible for a chronic purulent otitis media to exist for a considerable period without producing caries of the bony structures of the tympanum. For example, erosion of the superficial epithelial layer of the mucosa may be followed by the formation of firm granulations which protect the periosteum and underlying bone. When, however, the entire thickness of the tympanic lining membrane is at any point destroyed, the exposed bone, being deprived of its periosteal support, must depend chiefly upon its own arterial supply. Whether in such a case the denuded bone has been able to maintain its own nutrition, is usually indicated by the character of the granulations: *e.g.*, fairly rapid formation of a covering of firm, apparently healthy granulations usually indicates normal repair, whereas the slow formation of loose, friable granulations, through which a probe passes without appreciable resistance to bare bone, commonly indicates bone caries. This process involving the bony capsule of the labyrinth—*e.g.*, the promontory or the outer wall of the horizontal semicircular canal—may be self-limited, the diseased layer of surface bone being thrown off in the pus in the form of minute sequestra, and the bone being gradually covered by healthy granulations; or the necrotic process may involve the entire thickness of the labyrinthine wall, giving rise to a suppurative labyrinthitis. The latter result occurs in 1 per cent. of all cases of suppurative otitis media (Hinsberg). Destruction of the mucous membrane lining the vault, aditus, or antrum leads usually to a more destructive form of osseous disease, from which spontaneous local recovery is comparatively rare.

In the cellular bone surrounding the bony capsule of the labyrinth, and in that bounding the aditus and antrum, the disease in many cases involves a dual process, osseous necrosis or softening being apparently accompanied, or closely followed, by a proliferative osteitis, in consequence of which the diploic or pneumatic bone substance is ultimately replaced by very hard, compact bone. As a result of an extension of this process, the mastoid cortex is often greatly thickened, and the cellular character of the bone to a very considerable extent lost.

Much has been said as to the influence of this pathological osteosclerosis upon the gravity of the disease. Undoubtedly the marked increase in the thickness and density of the outer cortex renders an escape of pus through a perforation in this direction less likely to occur. On the other hand, it must be remembered that the process is not confined to the outer cortex, but occurs with equal regularity and frequency in the cellular bone surrounding the labyrinthine capsule, which it probably serves to protect. Bezold regarded it as a conservative process tending to limit the advance of a suppurative lesion in all directions.

Granulations; Polypi.—Whenever in the tympanum there is actual erosion or destruction of tissue, however superficial, the process of repair is inaugurated by the formation of new granulations, which may later become organized into firm fibrous tissue. The presence of a certain amount of firm granulation tissue points, therefore, not so much to the disease itself, as to the effort on the part of the organism to repair or replace that which has been

destroyed. On the other hand, the proliferation at certain points of loose, exuberant granulations, through which a probe at once passes to contact with rough denuded bone, is distinctly characteristic of osseous necrosis.

Aural polypi may assume almost any shape within the limits of the tympanum and auditory meatus. They may be single or multiple; may consist simply of masses of granulation tissue, or may become partially organized, covered by squamous or ciliated epithelium, and assume somewhat the character of fibromata. They may be attached by a comparatively broad base or by a very small pedicle. They may spring from any denuded surface,—*e.g.*, from the margins of a small perforation, from necrotic ossicles, from the promontory, or from the diseased tympanic margin, or tympanic ring. They are not infrequently attached to a denuded wall of the bony meatus near its tympanic margin. Very frequently they originate in the tympanic vault or the aditus.

In the author's experience, aural polypi point almost invariably to bone necrosis. While occasionally springing from the promontory, this site is exceedingly rare as compared with the outer wall of the vault or the diseased inner margin of the bony meatus. Aside from actual observation and experience, the comparative rarity of polypi springing from the promontory may be inferred from the rarity of suppurative involvement of the labyrinth as compared with the frequency of polypi as a manifestation of chronic middle-ear suppuration. That is to say, if any considerable proportion of the polypi observed in chronic purulent otitis media had their origin from a diseased promontory, one would expect more frequent suppurative invasions of the labyrinth than are known to occur.

Epidermization and Cholesteatoma.—A noticeable change in certain cases of chronic purulent otitis media is the conversion of the tympanic mucosa into a grayish-white, non-secreting membrane bearing some resemblance to the skin of the meatus. It is seen oftenest in association with marginal perforations involving Shrapnell's membrane and the upper posterior periphery of the membrana tensa. It may, however, occur with large central perforations. This so-called epidermization of the middle-ear cavity is inaugurated by extension of the epidermis of the meatus and drum membrane over the edges of the perforation and into the tympanic cavity. This process, by converting the recesses of the tympanum into dry cavities lined with a protective, non-secreting membrane, may be nature's method of limiting the spread of the disease. There can be no doubt that in many cases spontaneous recovery has resulted from this process (Schwartze).

Unfortunately, this happy sequence of events is not always realized. Let us imagine a case in which the epidermis of the canal, having entered the tympanum through a marginal perforation, extends by way of the aditus to the walls of the antrum. The new lining membrane, perhaps becoming macerated by pus from a deep-seated focus of suppuration, is ultimately exfoliated, leaving, however, a similar epithelial layer beneath. Successive layers, being similarly thrown off, finally become welded into a

rather compact mass which can not be expelled through the aditus, and must inevitably increase in size. Such a mass has received the name of cholesteatoma. Examined under the microscope, it usually contains numerous pus germs, some cholesterin crystals, and quantities of epithelial cells. The outer surface usually preserves the arrangement of concentric epithelial layers, which are easily separable one from the other.

The influence which cholesteatoma exerts upon the surrounding bone is one of gradual disintegration or absorption. The term absorption is in many cases more descriptive of the actual process than necrosis, since the latter destroys first the membranous lining or covering of the bone attacked. Cholesteatoma, on the other hand, may eat its way into contiguous bone, without the usual signs of suppuration, the enlarged and enlarging bone cavity retaining everywhere a well-defined epidermal covering. In such cases there may be little or no demonstrable discharge through the meatus, or there may be occasional very slight discharge consisting chiefly of the secretion from the newly-formed membrane mixed with decomposing epithelial cells. Such a discharge, when removed by means of a cotton-wound applicator, is usually unbearably offensive. In other cases the cholesteatomatous absorption of bone coexists, or rather alternates in different parts of the bone cavity, with the usual form of suppurative necrosis. The pus from such a lesion is often so indescribably offensive as to constitute in some degree a social barrier between the patient and his fellows.

Illustrations of these two types of the disease are found in two cases operated upon by the writer. In one, a man over fifty years of age, the radical operation was performed on account of some of the more commonplace symptoms of chronic middle-ear suppuration. On removing the cortex, the antrum, greatly enlarged, was found to contain a large cholesteatomatous mass, around which was a limited amount of secretion the stench of which was almost overpowering. In this case the operation was completed by the formation of a large meatal opening, and the patient made a perfect recovery. The second case was that of a strong Irish woman, a domestic, who suffered from a more or less constant discharge of so offensive a character that she found it difficult to retain any position longer than a few weeks. In this case cholesteatoma was found side by side with the usual signs of suppuration,—*i.e.*, pus, granulation tissue, and necrotic bone in adjacent spaces. Removal of these products of disease by the radical operation resulted in permanent cessation of the discharge. In both these cases, the evidence of progressive disintegration or absorption of bone rendered the danger of serious intracranial involvement, had surgical relief been withheld, very great.

The serious nature of these cases depends upon a fact now fortunately recognized by all experienced otologists,—viz., that cholesteatoma confined in parts of the ear not accessible through the external auditory meatus, may lead to dangerous spread of the infection,—*e.g.*, through the labyrinthine capsule, causing suppurative labyrinthitis; to the middle fossa of the skull, causing epidural or temporosphenoidal abscess; to the

posterior fossa, giving rise to infective sinus thrombosis or more rarely to cerebellar abscess.

Before leaving this phase of the pathology, it may be admissible to mention certain very exceptional cases in which the cholesteatomatous mass has caused such extensive destruction of the posterosuperior wall of the bony canal as to accomplish practically the mechanical results of a radical operation. Probably most aurists have seen one or more such cases. The writer some years ago had referred to him a patient whose aural lesion presented the following interesting features: The patient, a man of fifty years, had suffered since boyhood from occasional discharge from his right ear. Inspection revealed such extensive destruction of the posterior wall of the bony meatus that it was not difficult to remove from the antrum through the canal an epithelial mass the combined bulk of which equalled that of a chestnut. The cavity from which this mass was removed was apparently lined throughout with an epidermal covering which could easily be reached through the meatus. After removal of the growth, this patient was practically without aural symptoms up to the

FIG. 115.—Bone absorption resulting from cholesteatoma: *a*, cholesteatomatous mass; *b*, remains of canal roof; *c* spine of Henle; *d*, posterior canal wall.

time of his death from pneumonia some years later. Bezold was among those who recognized this condition as one reasonably insuring the patient's safety. The accompanying picture, drawn from a specimen in the writer's collection, illustrates very nicely a stage of the process by which this mechanical result is reached (Fig. 115).

Symptoms and Signs.—A suppurative process which by extension may reach the labyrinth, the brain cavity, or may infect the large venous channels of the brain, may obviously be the primary cause of many varied and complex phenomena. We shall limit our remarks, however, to a brief statement of the symptoms characterizing a suppurative lesion absolutely confined to the tympanum and adjoining mastoid cells. For the sake of brevity, it may be well to state in advance, that, aside from the discharge and the physical changes observed through the aural speculum, many patients endure this lesion for years without pronounced symptoms, either systemic or subjective.

While it would seem reasonable to assume that a lesion capable of maintaining a more or less constant purulent discharge might also cause considerable systemic disturbance, it is a clinical fact that constitutional symptoms are in most cases absent. This fact is so universally recognized among aurists, that the development of constitutional symptoms—*e.g.*, fever, pulse changes, etc.—would suggest to an experienced observer either an intracranial complication or some unrelated intercurrent disease. This does not necessarily imply that a chronic suppurative otitis media may not in some cases react unfavorably upon the patient's vitality, but simply that it rarely or never gives rise to characteristic symptoms of sepsis or of systemic disorder. The two symptoms of which these patients usually complain are the discharge and the impairment of hearing.

The Discharge.—The aural discharge varies greatly in different cases of chronic middle-ear suppuration, and is of some diagnostic significance. In the first place, the discharge has in the great majority of cases a distinctly offensive odor, whereas the discharge in acute purulent otitis media is usually odorless. It has been proved, however, that a mucopurulent secretion, unmixed with detritus resulting from necrotic bone or discarded epithelial elements, may be quite without odor. In certain neglected cases of chronic purulent otitis media, the exceedingly foul odor is due not so much to the tympanic changes as to the admixture with the tympanic secretion of decomposing epithelial cells from the diseased lining membrane of the bony canal. In such cases, measures applied to keeping the canal free of pus greatly modify the offensive character of the discharge. Chronic suppurative lesions characterized by deep-seated areas of bare and necrotic bone may give rise to moderate discharge of very offensive odor. In such cases, the secretion may be so limited in quantity that its odor becomes noticeable only when the aurist attempts to wipe out the tympanic cavity for purposes of examination. Finally there is a class of cases in which the foul character of the discharge may be a constant cause of discomfort and chagrin to the patient,—*i.e.*, cases in which the aditus and antrum are filled with cholesteatomatous masses, decomposing particles of which are constantly being washed out in the aural discharge. In these cases the peculiarly offensive nature of the discharge, and the presence in it of cholesterin crystals and quantities of broken-down epithelial cells, are of considerable diagnostic importance in determining the character of the lesion.

Physically the discharge may be thick or exceedingly thin; profuse or may consist merely of a few drops of viscid secretion confined to the recesses of the tympanic cavity, the presence of which may be unknown to the patient. The frequent or occasional presence of blood in the discharge points, as a rule, to the presence of polypi or exuberant, vascular granulations. Microscopic examination of the discharge in cases of chronic purulent otitis media practically always shows a mixed infection.

Deafness.—To some extent the hearing is impaired in almost every case of chronic suppurative otitis media. The functional loss depends on two conditions,—viz. (a) the partial or complete destruction of the membrana tensa and elimination of the malleus and incus from the function of sound transmission; and (b) hyperplastic changes in the region of the oval window interfering with the mobility of the stapes. Fortunately, complete loss of the drum membrane and also of the malleus and incus is by no means inconsistent with very good hearing,—*i.e.*, impairment so slight as to interfere but little or not at all with the patient's enjoyment of life. On the other hand, the formation of organized fibrous bands binding the crura of the stapes to the niche of the oval window may cause very disabling deafness. It is not always possible to determine the presence or extent of these hyperplastic changes by inspection alone. Thus, in two cases with extensive destruction of the membrana tensa and practical disarticulation of the ossicular chain by necrosis of the long arm of the incus, one patient may exhibit remarkable acuteness of hearing while the other may be exceedingly deaf. In each case the niche of the oval window may be filled with granulation tissue almost or quite hiding the stapes. In such contrasting cases, labyrinthine disease being excluded, we can explain the deafness of one patient as compared with the auditory acuteness of the other only by assuming that with the former fibrous adhesions between the stapes and the oval niche or window have taken place, while in the case of the latter the stapes is surrounded and covered by a mass of unorganized granulation tissue which acts vicariously with the stapes in the collection and transmission of sound waves from without. This hypothesis receives support from the numerous cases of chronic suppurative otitis media with retention of good hearing in which the hearing power has failed after spontaneous recovery and epidermization of the inner wall of the tympanum.

It is worthy of note that the deafness due to fibrous ankylosis of the stapes resulting from chronic middle-ear suppuration is regarded by Politzer as the type of deafness most amenable to treatment by surgical means, —*i.e.*, by division of adhesive bands passing between the head and crura of the stapes and the walls of the oval niche.

The impairment of hearing in these cases is of the type generally characterizing lesions of the sound-conducting mechanism. In those cases showing large perforations or practical destruction of the membrana tensa, the perception of the lower musical tones is always very considerably reduced. This is in accordance with the investigations of Helmholtz, corroborated by Bezold and others, which showed that the drum membrane is

particularly essential to the transmission of low musical tones. The middle and upper register may on the other hand be heard with apparently normal acuteness. In testing cases in which the sound waves were transmitted wholly by the stapes acting alone, the writer has met with a few instances in which there seemed to be some hyperacusis for the very high tones of the scale,—*e.g.*, the whistle. Hearing by bone conduction is regularly increased.

Physical Signs.—On attempting to inspect the ear by reflected light, the depths of the canal—*i.e.*, drum membrane or exposed inner wall of the tympanum—may be hidden from view by inflammatory products filling the auditory meatus. The canal may contain pus, detritus formed of pus mixed with exfoliated and decomposing epithelium, or may be occupied by a polyp partially or completely obstructing its lumen. The pus and epithelial débris are, of course, easily removed by the syringe or may be wiped out by means of a cotton-wound applicator. The removal of an aural polyp is not so simple a matter.

Large aural polypi—*i.e.*, polypi which nearly or completely fill the canal—are usually easily recognized at a glance. On the other hand, a small polyp, occupying the tympanic space or confined to the inner extremity of the bony meatus, may be somewhat puzzling to the student or beginner: he may find some difficulty in determining whether such a growth is a polyp, a swollen inner tympanic wall, or a bulging and raw, or denuded, drum membrane.

Diagnosis.—An aural polyp, large or small, presents the following physical characteristics: (1) It usually appears, as in most cases it is, nearer the orifice of the meatus, and therefore nearer the eye of the examiner, than either the bulging drum or the swollen tympanic mucosa. (2) It presents a raw and granular surface, usually easily distinguishable from the denuded drum membrane on the one hand and the swollen but intact tympanic mucosa on the other. (3) It is comparatively insensitive to palpation by the tympanic probe, provided the probe does not touch the canal wall, whereas the drum membrane and tympanic mucosa are extremely sensitive to pressure. (4) It is usually freely movable in the canal. (5) The probe, carried beyond the polyp, may be passed in all directions around it, except where it is attached to the wall of the bony meatus or to the tympanic ring, at which point the probe will be arrested. If the polyp is attached to some point on the inner wall of the atrium,—*e.g.*, the promontory (rare),—the probe will pass completely around it in either direction. In these manipulations great care must be observed to avoid bruising the polyp, or free hemorrhage is likely to occur, making further examination for the time being difficult or impossible. The question of removing the polypi through the canal, either for therapeutic purposes or to facilitate further examination, will be spoken of in connection with the treatment of chronic middle-ear suppuration.

The auditory canal being free of pus or other foreign matter, one is able to determine the position and size of the perforation. Only rarely is the drum membrane completely destroyed, the tough peripheral edge

(annulus tendinosus) being very resistant to suppurative disintegration. The majority of perforations are central apertures confined to the membrana tensa. They occur most frequently in the posterior segment (Figs. 116 and 117), but are also found in the anterior segment (Fig. 118). With large perforations of the anterior segment, the posterior margin, drawn inward by the hammer handle, is often adherent at the level of the umbo to the promontory (Fig. 119). Perforations involving both anterior and

Fig. 116.

Fig. 117.

Fig. 118.

Fig. 119.

Fig. 120.

Fig. 121.

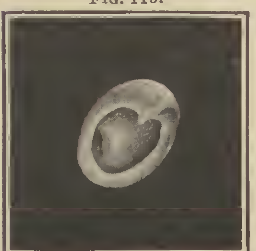

Fig. 122.

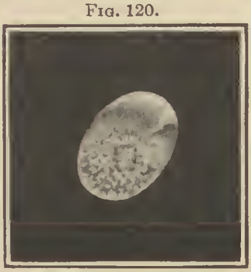

Fig. 123.

Fig. 124.

Central perforations.

posterior segments are not uncommon, and are seen chiefly in three forms, apparently representing different stages of the destructive process: viz., (a) the kidney-shaped perforation confined to the lower part of the membrana tensa (Fig. 120); (b) the horseshoe-shaped perforation involving large central portions of both anterior and posterior segments, the manubrium mallei remaining approximately intact (Fig. 121); and (c) the large irregularly heart-shaped perforation which is so often seen in cases of long-standing suppuration in which the hammer handle has been to a great

extent destroyed by the necrotic process (Fig. 122). All of the above examples fall under the head of central perforation when the peripheral edge is not at any point destroyed.

There is usually no great difficulty in mapping out the boundaries or limits of a central perforation so long as the surrounding surface of drum membrane is covered by normal or at least recognizable epidermis. The margins of a perforation of long standing are commonly thickened and easily distinguishable from the tympanic surface beyond.

A condition which is sometimes difficult to interpret correctly is found in cases in which the membrana tensa is not only perforated but also denuded of its epidermal covering, its outer surface being everywhere covered with fine granulations. When with this condition the mucosa covering the inner tympanic wall is swollen, granular, and crowded against the margins of the perforation, even the most experienced aurist may be unable to gauge correctly the extent of the injury until the remaining portion of the drum membrane has been restored by treatment to an approximately normal condition (Fig. 123).

The inner tympanic wall seen beyond the margins of a large central perforation may have the appearance of fairly normal mucous membrane, or may be so swollen that the normal elevations and depressions are to a great extent obliterated. Or, again, the mucosa may for the most part be replaced by granulation tissue (Fig. 124).

In some cases the lining membrane appears as a grayish-white, comparatively dry membrane having some resemblance to the skin of the canal. This change in the tympanic lining is brought about in part by the ingrowth through the perforation of the epidermal covering of the drumhead, and in part also by the action of the air upon the tympanic mucosa after the suppurative process as such has run its course. By this process, which is usually spoken of as epidermization, spontaneous recovery is brought about in certain cases.

Marginal Perforations.—It is clear that any central perforation, by extension of the destructive process, may be converted into a marginal perforation. The commonest and most characteristic marginal perforations are those in which the peripheral edge of the upper posterior segment of the membrana tensa and the adjacent part of Shrapnell's membrane are destroyed, leaving bare the bony ring to which they were attached (Fig. 125). In the majority of such cases, according to my experience, the adjacent portion of the bony frame is also diseased and very frequently to some extent destroyed. Thus, through a marginal perforation including and extending somewhat behind Shrapnell's membrane, one is occasionally able to see a considerable portion of the head of the malleus and body of the incus. This invariably indicates destruction of the inner, or tympanic, margin of the roof of the bony meatus (Fig. 126). With marginal, as with central, perforations, the landmarks of the tympanum may be completely hidden by granulation tissue or polypi (Fig. 127).

In marginal perforations exposing the normal structures of the vault,—

e.g., head of malleus or body of incus, or both,—these bones and the exposed tympanic cavity are seen in some cases to be covered by a white and apparently dry skin-like membrane. This condition has a different surgical significance according to the presence or absence of discharge. If there is, and for a considerable period has been, complete absence of discharge, it may represent the final stage of local resolution by the conversion of the vault recesses into dry skin-covered cavities (Fig. 128). On the other hand, a constant or even occasional flow of offensive pus from the aditus may be due to a cholesteatomatous collection within the antrum constituting a constant menace to life through its possible invasion of the labyrinth or intracranial cavity.

Perforations of Shrapnell's Membrane.—These little perforations seem pathologically and clinically to constitute a class by themselves. They

Fig. 129.

Fig. 125.

Fig. 126.

Fig. 130.

Fig. 127.

Fig. 128.

Marginal perforations.

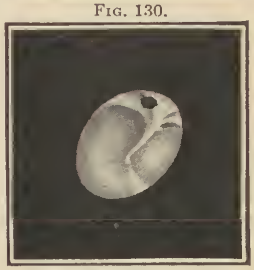

Fig. 131.

Perforations of Shrapnell's membrane.

are seen chiefly in three situations,—viz., immediately above the short process of the malleus (Fig. 129), above and a little in front of the short process (Fig. 130), and perforations extending backward through the posterior stria of Prussak (Fig. 131). While comparatively uncommon, a considerable number of these minute perforations can be seen during the year in any large aural clinic. The membrana tensa may be intact and apparently normal. Discharge from the vault may be fairly abundant, exceedingly slight, or wholly absent. Discharge being absent, there may

be a history of past tympanic suppuration. Quite frequently no such history is obtainable.

The exact pathogenesis of this condition has been, and still is, a subject of controversy. The statement of Dench[1] that "a perforation above the short process always means intratympanic caries, and usually indicates that the malleus is affected," is incorrect and might lead to surgical error. The ossicles may be, and in some cases certainly are, necrotic, but in many cases observed by the writer they were not.

Two facts must be taken into account in any attempt to determine the origin of these perforations, viz., (1) that they rarely occur during an acute middle-ear suppuration, and (2) their frequent presence in individuals whose ears present no evidences of tympanic suppuration either past or present.

Bezold stated that he had looked diligently for years for such a perforation occurring during acute suppurative otitis media, but had never seen one. He connected their origin etiologically with chronic obstructive lesions of the Eustachian tube, as a result of which Shrapnell's membrane is first retracted, then becomes markedly atrophic, and finally breaks down. This theory seems in part to have been held by Hartmann (quoted by Politzer), who stated that he had repeatedly observed the coincidence of a perforation of Shrapnell's membrane and a membrana tensa intact but retracted and adherent to the inner tympanic wall.

Politzer states that he has more than once seen the development of a perforation in Shrapnell's membrane during chronic middle-ear catarrh. His theory is that the membrana flaccida is first drawn into the depression above the processus brevis, where it subsequently breaks down under the influence of compression by epithelial masses, there accumulating from the skin of the auditory canal.

One of the most interesting contributions to the study of these minute perforations is found in a paper by Professor Schmiegelow,[2] published as far back as 1891. In it he described the physical changes in certain cases of acute suppurative otitis media in which a perforation of Shrapnell's membrane provided the only pathway for the escape of pus. Two of these cases recovered promptly under rational treatment, leaving, however, the perforation of Shrapnell's membrane as a permanent result. There is obviously no reason why such a perforation should be regarded as pointing to ossicular necrosis.

There are of course cases in which perforations of the membrana flaccida are clearly associated with ossicular necrosis.

Returning to the symptoms of chronic middle-ear suppuration, we must repeat the statement that, aside from the discharge, the impairment of hearing, and the physical changes in the ear itself, these patients are often practically without symptoms.

Pain is not usually present in chronic suppurative otitis media, except as betokening one or other of the following conditions,—viz., (a) retention

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[1] Dench: Diseases of the Ear, p. 399.

[2] Schmiegelow: Perforations of the Membrana Flaccida Shrapnelli, Arch. of Otology, vol. xx, No. 3, pp. 228–256.

of pus somewhere within the tympanic vault, aditus, or antrum; or (b) acute exacerbations of inflammation, usually involving the mastoid cells. As a rule, pus under pressure quickly finds a pathway of escape, with prompt relief of pain.

Many cases exhibit periodic variations in the amount of discharge, these changes being influenced by changes in the weather and other conditions causing nasopharyngeal congestion or inflammation.

Acute exacerbations, accompanied by mastoid tenderness, are always to be regarded seriously in cases of long-standing middle-ear suppuration, for the reason that an acute inflammatory process confined within bone spaces, which may have been the seat of extensive previous disease, may naturally be separated only by thin barriers from vitally important intracranial structures.

The course of the disease is very slowly progressive, and seems in many cases to be in some degree self-limited. That is to say, either by the process of osteosclerosis (eburnation) or by the formation of resisting walls of granulation tissue, the disease is in many cases confined during the major part of the patient's life to the antrotympanic cavity. This fact should be considered in its relation to the necessity for operative intervention. It does not, however, relieve the patient of the possibility of intracranial complication, which may ultimately occur in any case of chronic purulent otitis media.

Complications.—The complications of chronic purulent otitis media may be mentioned in the following order:

(a) Suppurative labyrinthitis, usually diffuse, rarely remaining circumscribed.

(b) Cerebellar abscess, infection very frequently by way of the labyrinth.

(c) Cerebral abscess.

(d) Meningitis, most often accompanying some suppurative lesion of the brain.

(e) Infection of the sigmoid (lateral) sinus.

There is no longer any doubt, that, in a very large percentage of cases of intracranial disease of otitic origin, the infection of the brain or meninges is secondary to suppurative labyrinthitis. That this relation has not been more commonly recognized is due to the fact that the phenomena of labyrinthine disease have only recently received adequate recognition. Undoubtedly many cases of intracranial suppuration have been operated upon, and ended fatally, without any suspicion on the part of the medical attendants of a pre-existing suppurative labyrinthitis.

Treatment.—The treatment of chronic purulent otitis media consists essentially of local treatment of the various morbid changes present, and in no disease do the physical changes vary so widely. Polypi, if present, must be removed, either through the canal or by radical operation; ossicles, if to a certain extent diseased, must be removed; cholesteatoma must be dealt with radically. The ear should be irrigated in some cases, whereas in others irrigation is contra-indicated. Finally, there are certain cases which, in the author's opinion, call for little treatment other

than certain simple measures for cleansing the ear, in which the patient should be instructed. Obviously no routine method of treatment can properly be described for this disease. For example, routine irrigation of the ears with antiseptic solutions does not assure surgical cleanliness and rarely results in recovery. Clearly we must classify our cases before we can suggest rational lines of treatment.

I. Let us consider first the simplest form of the disease,—viz., a case of comparatively short duration,—*i.e.*, not over one or two years,—and presenting the following conditions: central perforation of small or medium size which refuses to heal; discharge persistent but moderate in amount and free from the offensive odor characteristic of bone necrosis. Such a lesion falls under the head of chronic purulent otitis media when it has passed the stage in which spontaneous recovery may be hoped for.

In searching for the chief obstacle to local recovery in a case of this character, we may find it in one or other of the following conditions: (a) In the first place, the mere presence of a small perforation subjects the middle ear to constant reinfection from the nasopharynx. With an intact drum membrane, occasional condensation of the air in the nasopharynx does not affect the tympanum further than to produce temporary increase of intratympanic pressure. With a perforation, on the other hand, every such condensation, as when the patient sneezes or blows his nose, carries a stream of air, impregnated with the impurities of the nasopharynx, through the tympanum. (b) The epidermal covering of the drum membrane may have extended over the edges of the perforation, rendering its closure by cicatricial tissue no longer possible; and (c) the lower part of the tympanic cavity may contain residual pus which the perforation by reason of its position can not drain. Routine irrigation with any solution is without value in such cases, and not infrequently is distinctly injurious.

Bone disease being excluded by the character of the discharge, the treatment should aim at protection of the tympanum by closure of the perforation. As an initial measure, the canal should be wiped free of pus and dried. The nose and nasopharynx should be sprayed with some mild cleansing solution, the ear carefully inflated per catheter, and the drum membrane reinspected. Ordinarily a certain amount of pus or mucus will have been blown out into the canal, giving some idea of the condition of the lower (hypotympanic) space. The canal and drum membrane are again wiped out, swabbed with alcohol, and the canal lightly packed with a wick of sterile gauze. This is to remain in the canal until the patient is seen by the aurist on the following day. This treatment should be repeated for several consecutive days, when we shall have gained fairly accurate knowledge of the tympanic condition. If the discharge is very slight, so that the canal and wick are found practically clean at the daily dressing, we may proceed at once in our efforts to close the perforation. If, on the other hand, a considerable amount of secretion is discharged daily, it may be necessary to obtain better drainage by a free incision through the membrana tensa, after which the wick treatment is continued as before.

When the discharge is reduced to a minimum, it may be found that the perforation is gradually closing under the stimulation of the daily cleansing and inflation. If no tendency to closure is observed, it may be that this is prevented by epidermization of the edges of the perforation. To correct this the edge of the perforation should be very lightly cauterized. For this purpose trichloracetic acid is the best agent. It is applied in the following way: The drum membrane is cleansed and thoroughly dried. A fine-pointed cotton applicator is tightly wound with a minute amount of absorbent cotton, which is dipped in sterile water, the excess of which is removed by sterile gauze. The cotton-wound end of the applicator is then plunged into a bottle containing crystals of trichloracetic acid, and rotated several times. Any adherent crystals are removed. Under inspection by reflected light, the edges of the perforation are very lightly touched with the applicator. The influence of the acid is seen at once in slight whitening of the edges. The perforation may then be closed by a disk of paper prepared as follows: A disk of thin paper is cut of a size to extend slightly beyond the edges of the perforation; this is soaked in 95 per cent. alcohol, and then carried to its position covering the perforation by means of tympanic forceps or on the point of a cotton-wound applicator. Such a disk, if properly applied, adheres closely to the drum membrane. If no inflammatory reaction results, it should be allowed to remain in position until carried outward to the canal wall by the outward growth of epidermis covering the membrana tensa. Quite frequently the perforation will be found greatly reduced in size or may be closed. This, by protecting the tympanic mucosa, may end the lesion as a suppurative process.

In some cases this treatment may have to be repeated several times before the perforation is closed. In still other cases it may be impossible to obtain complete closure of the perforation, the dry, cleansing treatment resulting, however, in cessation of discharge. While a perforation persists, however, the patient remains subject to recurrences through infection from without, or from the air current from the nasopharynx.

II. *Large Central Perforations without Polypi or Excessive Production of Granulation Tissue.*—It will be remembered that a central perforation may mean destruction of the entire membrana tensa with the exception of the marginal or peripheral edge (annulus tendinosus). In this condition restoration of the drum membrane is no longer possible.

Under this head I wish to refer briefly to a class of cases with which all aurists are probably familiar, but which have, as a rule, received scant attention in otological literature. The physical picture is somewhat as follows: There is extensive destruction of the membrana tensa and frequently also of the hammer handle, the remnant of which may be adherent to the promontory. The tympanic walls are covered with mucous membrane or a layer of firm granulation tissue which does not disguise the landmarks of the inner tympanic wall,—*e.g.*, promontory, niche of round window, etc. (Figs. 117 and 122). Rough, denuded bone is nowhere demonstrable either by inspection or by the tympanic probe. The dis-

charge or tympanic secretion may be slight, or may appear abundant simply as a result of neglect. In the latter case rational cleansing treatment soon reduces its amount. There are no evidences of labyrinthine involvement. Hearing tests may demonstrate a very fair degree of hearing power, the patient being at no disadvantage in communicating with his fellows; or the hearing may of course show considerable impairment.

How shall we interpret this condition for ourselves and to our patients? Personally I believe that it in many cases represents a suppurative process which has run its course, or at least has become stationary, having erected its own barriers, either in the condensation of the bone itself, or in the production of firm granulation tissue between the focus of disease and surrounding structures.

So long as the tympanum is lined by mucous membrane or granulation tissue, there must of necessity be some secretion or discharge, though this may be very limited in amount. This discharge can be absolutely terminated only by converting the tympanic spaces into skin-lined cavities. But we can not be sure that such epidermization may not result in very considerable reduction of the patient's hearing power. Every aurist knows of cases in which this has occurred.

The point I wish to make is this: We should not be slaves to the dogma, too often repeated in otological literature, that aural discharge is necessarily of itself a danger signal, or one which when persistent calls for operative intervention. If we can satisfy ourselves that the discharge is simply the logical product of exposed mucous membrane or healthy granulation tissue, and that there is no evidence of an active suppurative process involving the bone itself, we should not be too ready to interfere with nature's method of conservation.

Treatment.—In my experience, the condition just described is not, as a rule, improved by routine, frequent irrigations. If when first seen the canal and tympanum show the result of neglect in residual pus,—usually very offensive in odor,—this should be removed by means of sterile cotton on applicators. The canal should next be filled with hydrogen peroxide, which should be allowed to remain at least five minutes. The ear is then syringed with a warm solution of boric acid, after which the canal is filled with 95 per cent. alcohol, which in turn is allowed to remain a few minutes. The alcohol, itself an efficient cleansing agent, by its quick evaporation leaves the canal comparatively dry. Finally the canal and tympanum are "dusted" with boric-acid powder, introduced by a powder-blower. The canal is lightly filled with sterile gauze or the concha filled with sterile cotton. The patient is instructed to return at regular stated intervals for observation, at first on consecutive days. This treatment has the undoubted advantage of reducing the amount of the discharge.

When, finally, the physician has through careful treatment reduced the amount of discharge to a minimum, the patient should be instructed in keeping the ear clean. Many cases do well on the following routine treatment: Regularly once a week, the patient is to cleanse the ear by the

method first described,—using first hydrogen peroxide, then thorough boric-acid irrigation, and lastly alcohol. Following this the ear is to be dried with sterile absorbent cotton. While out of doors, the ear should be protected by a bit of sterile cotton in the concha, but not introduced into the meatus. Once daily, preferably at night, the meatus should be filled with an alcoholic solution of boric acid (gr. xx ad ℥j), which is retained in the ear a few moments only. If the alcohol causes severe "burning," it may be diluted with sterile water, half and half. Under this treatment many patients suffer little or no discomfort, and the lesion remains quiescent or its character may finally be changed by epidermization of the tympanic cavity. In some cases the frequency of the irrigations may be reduced to once in ten days; in others its repetition every three or four days may be required. It should, however, be repeated no oftener than is necessary to free the canal and hypotympanic space of residual pus.

It is, of course, not claimed that the treatment above outlined will prove successful in every case presenting the physical characteristics described. There are some cases, for example, in which the discharge, whether it be scant or profuse, is perpetuated by a diseased condition at the tympanic mouth of the Eustachian tube. The tubal lesion may consist of an area of superficial osseous necrosis easily within the reach of a

FIG. 132.—Yankauer's Eustachian curette.

curette through the meatus; or, again, one aspect of the tube may be denuded of its mucous lining while a persistent mucosa of the opposite wall prevents the fibrous closure of the tube which is the first step toward permanent cessation of the discharge. When it can be determined that the principal seat of disease is located here, thorough curettage of the tubal region may bring about a cure. The purpose should be not only to remove any small focus of necrotic bone, but absolutely to remove the mucosa from that portion of the canal curetted. Possibly the best method of accomplishing this end may be that proposed by Yankauer. This procedure is carried out by means of a specially devised curette in the following way: The tympanic cavity, including both atrium and vault, is very carefully cleansed. The Eustachian canal is cocainized by means of a 10 per cent. solution of cocaine, carried through the tube first by a cotton-wound applicator and then introduced through the Eustachian catheter. The tympanum and pharyngeal end of the tube are also cocainized. The Eustachian curette (Fig. 132) presents a shaft so curved that it may be introduced through the meatus and tympanum into the tube as far as the isthmus or slightly beyond. The cutting part consists of a terminal disk attached centrally to the end of the shaft. When carried to its position in the tube at or just beyond the isthmus, the shaft is rotated slightly back and forth so that a circular cut through the mucous membrane is

made. When the surgeon is satisfied that the mucosa is completely divided throughout the entire circumference of the canal, the instrument is sharply withdrawn, separating the mucosa from the bony canal, and in some cases bringing it out inverted into the tympanum. This in favorable cases induces the formation of healthy granulations and subsequent permanent closure of the tube. When this result is obtained, discharge from the tube naturally ceases, and the tympanum is no longer subjected to this source of reinfection.

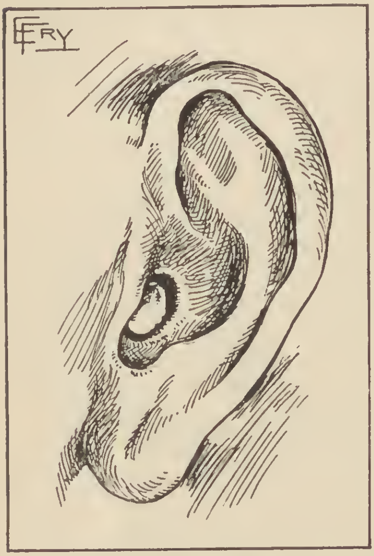

FIG. 133.—Aural polyp.

III. *Aural Polypi and Granulations* (Fig. 133).—Polypi occluding the auditory canal interfere not only with drainage but also with any plan of treatment. Polypi must therefore be removed either through the canal or by more radical operation. The first step is the determination of the point of attachment by means of the probe.

Most polypi are attached to the tympanic ring, the posterior wall of the bony meatus, or spring from some point in the aditus. In these situations, they can usually be removed either in part or whole by means of the snare or a sharp ring curette, the latter instrument being carried beyond the polyp and pressed against the bone surface to which the pedicle is attached. Unfortunately, such removal is very often followed by rapid re-formation of the growth.

As a comparatively rare condition, polypi of considerable size have been found depending from the edges of a small perforation in the drum membrane. In such a case, the careful use of the probe detects the surrounding membrana tensa. Removal of the polyp in such a case has resulted in a cure (Bezold).

Polypi springing from the promontory or other part of the inner tympanic wall are rare as compared with the marginal attachments above referred to, and also represent a more serious condition. It must be remembered that the appearance of a polyp springing from any bone surface invariably represents nature's effort to limit a necrotic process at that point. The attempt to remove a polyp springing from the promontory or the region of the oval window through the external auditory canal may be mechanically successful, but may too easily give rise to a labyrinthine fistula, and consequent suppurative labyrinthitis, to appeal to me as good surgery. I am inclined, therefore, to regard polypi attached to the inner tympanic wall—the labyrinth itself having escaped invasion—as valid and sufficient grounds for performing a radical operation.

Polypi not being present, or having been removed, the landmarks of the tympanum may be obscured or hidden by exuberant granulations (Fig. 124). This condition may be largely the result of neglect, the probe finding everywhere beneath the granulations a firm bed of healthy tissue, or, in other words, an absence of bare, necrotic bone. In this case, careful cleansing treatment, with occasional judicious use of astringents,—*e.g.*, nitrate of silver, preferably in the form of a bead fused upon the end of an applicator,—may soon bring about a healthier condition. I have seen cases, in which the granulations were apparently exuberant and the discharge exceedingly offensive, show marked and quick improvement as a result of the following plan of treatment: The ear having been cleansed by the usual method and dried, boric-acid powder in considerable quantity is introduced into the tympanum, not by a powder-blower, but by means of a spatula. With a probe or other blunt instrument, the powder is well distributed over the tympanic surface, a central space being made for a sterile gauze wick. The wick is packed rather firmly in the tympanic cavity and loosely in the meatus. It is a mistake to believe that boric acid thus used may act as a dangerous obstruction to the flow of pus. Some of the powder is apparently always absorbed, and pus escapes through or around the remainder and is taken up by the gauze wick. The action of boric-acid powder thus directly applied is usually to cleanse and deodorize the cavity of the tympanum, and also in many cases to cause noticeable shrinkage of the granulations. In other words, it is both disinfectant and astringent.

In other cases the probe quickly reveals a lesion of different character, —passing without sense of resistance through the granulations to bare, necrotic bone. In this condition I can see no safe method of treatment short of a radical operation.

IV. *Marginal Perforations.*—In the great majority of cases marginal perforations are accompanied by evidences of bone necrosis. Nearly always the contiguous portion of the tympanic ring is diseased, and the tympanic end of the roof of the bony meatus is occasionally eroded to a very considerable extent. In the latter instance the tympanic vault may be open to inspection, and the head of the malleus or body of the incus brought into plain view (Fig. 126). When the posterosuperior canal wall is to a considerable degree destroyed, we are frequently able to introduce a bent tympanic probe backward and slightly upward into the aditus and antrum. This condition, while giving evidence of the destructive power of the lesion, frequently adds to the patient's safety by providing free drainage from the antrum and upper mastoid cells. Another frequent site of osseous necrosis is found in the ossicular chain. Almost invariably with large marginal perforations involving both membrana tensa and membrana flaccida, the malleus and incus will be found diseased. They may be completely destroyed. The incus, from its position in relation to the aditus and antrum and from its poorer blood supply, is the most frequent victim of the necrotic process, the malleus coming next. The stapes, by

some fortunate provision of nature,—probably its abundant blood supply through the annular ligament,—is only rarely involved. The writer recalls at least three cases, however, upon which he performed a radical operation, in which no vestige of any ossicle, malleus, incus, or stapes, was present. In two of the three cases the labyrinth was involved in the suppurative process; in one the labyrinth was not involved.

The treatment indicated varies somewhat in accordance with the following conditions,—viz., (A) The presence of bare necrotic bone, (B) evidence of pus retention in the vault, (C) necrosis of the head of the malleus and body of the incus, and (D) recurring polypi or exuberant granulations in the aditus and vault.

A. Osseous necrosis is evidenced in some cases by the constant recurrence of exuberant granulations at some point, in others merely by the presence of bare rough bone. The significance of an area of necrotic bone as a surgical indication varies with the region involved. Thus, in surface necrosis of the promontory or region of the oval window, the danger of labyrinthine fistula and suppurative labyrinthitis calls for surgical intervention. On the other hand, a large marginal perforation involving both membrana tensa and Shrapnell's membrane, and causing destruction of the upper posterior segment of the bony ring, may, by providing free drainage from the vault, aditus, and antrum, render the condition much less dangerous (Fig. 134). When with this condition the promontory is not diseased, and the antro-attic cavity is not occluded by polypi, a bent probe passing without resistance backward and upward into the aditus and antrum, it is usually possible to cleanse the whole antrotympanic cavity by the measures already advocated in the treatment of large central perforations. Such a case—particularly if functional tests demonstrate fairly good hearing power—should, in my opinion, be subjected to careful and rather prolonged cleansing treatment before a radical operation is decided upon.

B. Another variety of the lesion, by no means uncommon, is one in which, with extensive destruction of the membrana tensa below the posterior fold, that portion above the posterior fold and also Shrapnell's membrane remain intact. The lower margin of this remnant of membrane, being more or less indrawn and adherent to contiguous structures, gives rise to occasional pus retention within the vault (Fig. 135). These patients suffer from periodic attacks of earache,—during which antrum tenderness is usually demonstrable,—these acute symptoms being usually relieved only after a free flow of pus from the vault is re-established.

I believe that this is one of the most dangerous forms of the disease. An incision through the bulging remnant of membrane, the knife being carried upward into the vault, relieves the pain of the acute attack, but does not provide against recurrences. When operated upon radically, the aditus and antrum, and frequently the mastoid cells, are found to be extensively diseased. In the writer's opinion, these cases represent the type of lesion in which extension of the suppurative process to intracranial

structures is most likely ultimately to occur. The safest treatment lies, therefore, in surgical intervention,—*i.e.*, the radical operation.

C. *Necrosis of the Malleus and Incus.*—Necrosis of the bodies of the malleus and incus is usually accompanied by the presence of surrounding polypi or exuberant granulations. The ossicles themselves may, therefore, be hidden from view, even when the attic is exposed by erosion of the tympanic end of the roof of the bony meatus. The ossicles may be only slightly eroded or may be to a very great extent destroyed. In this condition granulations springing from the diseased ossicles, and also from the adjacent walls of the attic and aditus, may interfere very seriously with drainage of pus from the antrum (Fig. 136).

Fig. 134.

Fig. 135.

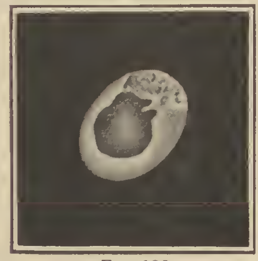

Fig. 136.

Fig. 137.

Perforations of long standing.

D. A somewhat kindred condition is that in which, evidences of ossicular necrosis being absent, the vault is filled with unhealthy granulations, apparently springing from the walls of the aditus (Fig. 137).

The treatment of these conditions by any means short of surgical intervention requires patience and carries with it prolonged uncertainty as to whether an operation will not in the end become necessary. When the disease is perpetuated chiefly by ossicular necrosis, the logical means of relief would seem to be surgical,—*i.e.*, either the removal of the ossicles through the auditory canal, or the more complete removal of all diseased structures by the radical operation. The relative value of these operations will be referred to later.

When the chief obstacle to drainage is found in polypi or exuberant granulations springing from the walls of the aditus, and a large marginal

perforation brings this region into the practical reach of instruments, an attempt may be made to remove them through the canal, though I believe it is better surgery, and will prove in the majority of cases to the advantage of the patient, to proceed at once to the radical operation.

Technic.—To begin with, the ear should be irrigated with bichloride of mercury solution (1 in 4000), and dried. A gauze wick, saturated with 10 per cent. solution of cocaine, should be packed rather tightly against, and if practicable behind, the granulations and allowed to remain some ten minutes. We may then try to remove the granulations with a sharp curette. The ring of the curette should be carried in front of and beyond the granulations, and an effort made to remove them with one stroke of the instrument. With the cutting edge directed backward and outward against the inner surface of the tympanic ring, no damage can be done, whereas pressure of instruments inward against the inner tympanic wall is fraught with dangers outweighing in importance the advantages to be gained. Even after the use of cocaine, the first stroke of the curette is usually followed by free hemorrhage, interfering with the further exact use of instruments,—unless much time be allowed for repeated application of adrenalin, cocaine, etc. Enough may be removed, however, to provide drainage of the attic and spaces behind. Following this curettage, the wound is again irrigated, dried, and lightly packed with sterile gauze. For some days thereafter the patient should be seen daily. One of the best dressings in these cases is by a fairly thick covering of boric-acid powder, against which a wick of sterile gauze is lightly packed. This dressing should at first be changed daily, the ear being cleansed and dried each day before the wick is replaced. Under this treatment the tympanic condition may change markedly and fairly rapidly for the better. When finally the use of wicks is discontinued, we should adopt as nearly as possible a dry method of treatment, frequent irrigation with warm solutions unquestionably favoring the re-formation of redundant granulations. Should the granulations re-form within the aditus, it will usually be better to resort to the radical operation.

V. *Perforations of Shrapnell's Membrane.*—Perforations confined to Shrapnell's membrane may for practical purposes be divided into two classes, viz., (1) those associated with a suppurative process within Prussak's space, the tympanic vault, or even in the aditus or antrum beyond; and (2) those in which all signs of tympanic suppuration are absent. The writer personally believes that these small perforations without any evidences of present suppuration represent in many cases the result of a past infection of one of the membranous spaces of the vault; and further that such lesions, if circumscribed, may be credited with a certain tendency toward spontaneous resolution. This hypothesis seems justified by the number of such cases (Figs. 129, 130 and 131) in which, with a history of acute tympanic disease long since ended, discharge is absent or negligible, and the hearing but little or not at all impaired. These little perforations are in many cases so inconspicuous as to be easily overlooked unless systematic search is made for them.

When the discharge is continuous and profuse, one may be obliged to infer that the attic, and probably also the aditus and antrum beyond, are involved in the suppurative process.

Treatment.—When the hearing is good and the discharge very slight, I am inclined to let these ears very much alone. Occasional irrigation by the physician himself by means of the attic syringe or cannula may be of value. I believe that this should be repeated only when there is evidence of pus collection behind the perforation. The use of alcohol, or alcoholic solution of boric acid, the canal being partly filled therewith once or twice daily, is of distinct value in some cases.

When the discharge is so profuse as to constitute a proof of extensive suppurative involvement of the vault, surgical intervention may be called for. In this condition, I am inclined to believe that a radical operation is in many cases not indicated, and that a rational procedure, and one which does not endanger the hearing power, is by a simple mastoidectomy, the aditus being very carefully and thoroughly cleared of granulations and diseased bone. The aditus should be kept open until all evidences of attic suppuration have disappeared. I have used this method in a few cases of long-standing attic suppuration with very good results.

Obviously there will be cases in which this operation will fail on account of necrotic changes requiring more radical treatment than a simple mastoidectomy makes possible. When the simpler operation proves successful, the gain to the patient in the saving of drum membrane and ossicles and the preservation of function is very great. For this reason, the simpler operation is not only justified, but, in my opinion, indicated in cases in which it offers fair promise of success. The patient, however, should be forewarned as to the possible necessity of later recourse to the radical operation.

Cholesteatoma.—The presence of cholesteatoma within the antrum can not always be determined. In some cases, however, characteristic masses may be seen projecting into the vault from the aditus, and in others their presence may be inferred from the persistently offensive character of the discharge or the occasional presence therein of characteristic epithelial elements. Systematic examination of the discharge under the microscope for cholesterin crystals and broken-down epithelial cells would probably indicate its presence in many unsuspected cases.

The treatment of cases in which the presence of cholesteatoma can be determined may be summed up in three words,—*the radical operation.* Such a growth, from its power of causing osseous disintegration or absorption in any or all directions, constitutes a condition in which the patient is constantly exposed to the danger of intracranial infection. The operation in these cases should provide for an enlarged meatal opening through which all parts of the tympanomastoid cavity can be reached, and subsequent cholesteatomatous collections prevented.

Operative Indications.—From a descriptive view-point it is clear that "chronic purulent otitis media" is an elastic term covering a variety of conditions. While some of these constitute a positive menace to the life

of the patient, in others the danger of intracranial invasion is exceedingly remote. The responsibility of the surgeon in the matter of his advice to the patient is, therefore, not small. If his duty in some cases is to state positively his belief that the patient's safety demands surgical intervention, there are others in which it is no less his duty to state with equal frankness his belief that operation is not required, and this without emphasis upon remote eventualities which the patient is not likely to experience.

In this connection I am reminded of a remark of Professor Körner of Rostock, in watching whose work I had the privilege of spending a very instructive week some years ago. Upon my expressing the hope that I might see him perform a radical operation, he said: "Well, we may have to wait for the proper case. There are so many cases of chronic purulent otitis media, but so few in which the radical operation is really indicated."

The radical operation is an attempt at once to remove all diseased bone and to provide absolutely free drainage of all infected recesses of the antro-tympanic cavity by way of the external auditory meatus.[3] Many attempts have been made to formulate the indications for this procedure, resulting in some cases in lists of considerable length. In the writer's opinion there are comparatively few conditions which can safely be given as positive indications for this operation. These may be mentioned somewhat in the following order:

1. The persistent recurrence of exacerbations of acute inflammation accompanied by mastoid tenderness or pain and physical evidences of pus retention in the vault. In these cases we may assume that the antrum and mastoid cells are the seat of a subacute suppurative process, an advance of which may be recorded with each succeeding attack. I believe that these recurrent attacks indicate one of the most dangerous forms of the disease, and that they should be regarded as a positive indication for the radical operation.

2. Evidences of fistula leading from the mastoid cells either through the posterior wall of the bony meatus or through the outer cortex. The latter condition gives rise either to marked postauricular œdema or to subperiosteal abscess. Mygind,[4] analyzing 100 cases of subperiosteal abscess treated in the Copenhagen Commune Hospital, observed 22 cases in which the cortical perforation resulted from an acute mastoid inflammation complicating chronic suppurative otitis media. In 16 of these cases (75 per cent.) the vault and mastoid cells were filled with cholesteatoma. This would apparently indicate that postauricular swelling points usually to a more serious condition in chronic middle-ear suppuration than in primary acute disease of the tympanum and mastoid. It is a positive indication for immediate surgical intervention.

3. The dangers of cholesteatoma within the antrum or mastoid cells have already been explained. Hinsberg, Neumann, and others who have

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[3] The radical operation is described in detail in the section devoted to Aural Surgery.

[4] Mygind: Subperiosteal Abscess of the Mastoid Region, Annals of Otology, 1910, pp. 529–540.

studied the etiology of suppurative labyrinthitis, have found that a large percentage of cases are traceable to the action of cholesteatoma upon the bony capsule of the labyrinth. The determination of cholesteatoma is, therefore, a positive indication for the radical operation.

4. Polypi springing from the promontory or region of the oval window can not be removed through the auditory meatus without great danger of producing a labyrinthine fistula. They are, therefore, an indication for a very careful radical operation.

5. Persistently recurring polypi from any part of the tympanum—showing bone necrosis which non-operative treatment fails to eradicate—constitutes a logical reason for performing the radical operation.

6. Very profuse and persistent discharge,—either by reason of the patient's discomfort or the evidences it may afford of deep-seated bone disease, may constitute a valid reason for surgical intervention.

7. Facial paresis or paralysis, occurring during the course of chronic middle-ear suppuration, evidences of suppurative labyrinthitis being absent, is a positive indication for very careful performance of the radical operation.

If in the above list the author has omitted certain conditions usually included among the indications, he has done so with intention. For example, one writer, for whom the author entertains the greatest respect, includes "recurrent attacks of dizziness, nausea or headaches" occurring in chronic purulent otitis media as the operative indications. These symptoms may or may not call for the radical operation. If due, for example, to suppurative labyrinthitis, the radical operation alone would be most positively contraindicated. This brings us to the consideration of a rule which all aural surgeons will do well to respect,—viz.: *No patient should be subjected to the radical operation until his labyrinth has been carefully tested for evidences of suppurative labyrinthitis.*[5]

Ossiculectomy.—Before leaving the subject of treatment, a word should be said of the operation of ossiculectomy. This operation proposes to provide freer drainage from the vault, aditus, and antrum by the removal of the diseased malleus and incus and incidentally of diseased remnants of the drum membrane which might interfere with the escape of pus from the vault. It is, therefore, in a way a substitute for the radical operation. If it were possible to determine that the diseased ossicles were the sole obstacles to resolution, or that their removal would insure resolutions in spite of infection within the mastoid cells, the arguments in favor of this procedure would have a more logical basis. I know, however, of no positive indication for the selection of this operation, for the reason that there is no way of positively excluding deep-seated foci of bone disease which also might require surgical eradication. For this reason I have personally preferred the radical operation in all cases of

[5] The various tests for suppurative disease of the labyrinth are given in the chapter dealing with Suppurative Labyrinthitis.

chronic purulent otitis media in which surgical intervention was deemed necessary. I can, however, conceive of many cases in which the slighter operation might prove successful, and in this event the advantages to the patient might be very considerable.

The most favorable cases for the operation are those in which, with evidences of ossicular necrosis, there is no demonstrable exposure of bare bone in other directions, and in which the patient has retained good hearing power.

In such a case I see no objection to this operation, the patient being made to understand that the results are somewhat uncertain and that in case of failure a radical operation may later become necessary.

CHAPTER IX.

CHRONIC NON-SUPPURATIVE DISEASES OF THE MIDDLE EAR; OTOSCLEROSIS.

Chronic catarrhal otitis media is an elastic term rather indefinitely applied to a variety of tympanic conditions not easily classified. There can be little doubt that many of the conditions resulting in impaired hearing, and described in text-books under different names as pathological entities, are in reality but different stages of a slowly progressive lesion. Our knowledge of these conditions is based very largely upon clinical study of cases, and observation of such morbid changes as can be seen in the living patient, and only to a limited extent upon actual study under the microscope of the tissue changes involved. It is clear, therefore, that, whatever classification may be adopted, the different groups will be found more or less to merge one into the other, and it is by no means impossible that future investigation may show more definitely either their pathological relationship or independence.

The chronic non-suppurative lesions of the ear may be considered under the following heads:

1. *Chronic tubal catarrh* without marked tympanic changes other than those due to retraction of the drum membrane.
2. *Chronic catarrhal otitis media.*
3. *Chronic hyperplastic otitis media;* dry catarrh of the middle ear.
4. *Otosclerosis.*

CHRONIC TUBAL CATARRH.

Obviously a catarrhal condition of the Eustachian tube may exist for a considerable period without producing ear symptoms. Only those cases, therefore, are likely to come under the otologist's care in which the calibre of the tube is so reduced as to interfere with the passage of air to the tympanum.

The causes of chronic tubal catarrh are exactly the same as those mentioned as predisposing factors to the acute type, and need not be repeated here. In nearly every case will be found some condition either in the nose or nasopharynx interfering with nasal respiration.

The history in characteristic cases is that of very moderate impairment of hearing characterized by periods of amelioration. The patient may tell you that there are considerable periods during which he is not conscious of any loss of auditory acuteness; or he may associate his periods of improvement with periodic changes of residence,—*e.g.*, as related to vacation periods in the mountains or at the sea-shore. Usually the hearing is very considerably influenced by weather conditions, improving in dry clear weather and relapsing with the reverse conditions. With the

impairment of hearing, and often changing with its variations, tinnitus aurium is almost invariably present. Interrogation usually elicits the fact that the patient suffers at times from difficulty in nasal respiration, and physical evidences of some degree of nasal obstruction are usually present.

Functional examination shows very moderate impairment of hearing of the general type characteristic of disease confined to the conducting apparatus,—*e.g.*, slight loss of hearing for the lower musical tones and some increase in hearing by bone conduction. The ratio between hearing by air conduction and bone conduction is never reversed in a chronic lesion confined to the Eustachian canal.

On physical examination the drum membrane is found to be retracted. In adults suffering from chronic tubal catarrh of not very long standing, the retraction may be of moderate grade, more pronounced displacement being at first opposed by the natural strength and resistance of the membrana tensa. Children, on the other hand, not infrequently exhibit with chronic tubal disease very extreme grades of retraction.

A very characteristic feature of chronic tubal catarrh is found in the fact that the patient's sense of functional loss or disturbance is frequently out of all proportion to any functional changes which may be demonstrated by the usual hearing tests. Conversational and whispered speech and the acoumeter may be heard at distances only moderately under the normal, and the change in the lower tone limit may be so slight as to leave one in doubt as to its significance. If the lesion be unilateral, however, there is a demonstrable difference—*i.e.*, increase—in bone conduction in the involved ear. As compared with these rather negative findings, the patient himself is in no doubt as to his sense of diminished hearing power. I believe that a recognition of this seeming paradox or contradiction is extremely important, for otherwise we may believe, and assure the patient, that his auditory disturbance is transitory and of little importance, whereas his aural disorder is in reality at just that stage requiring most careful study and attention in order to avert changes leading to later serious functional loss.

Another characteristic feature of this lesion is the very marked subjective sense of functional improvement resulting from inflation. The impairment, it must be remembered, is a mechanical one, due, in the early stages of the disease, not to pathologic changes within the middle ear, but to partial fixation of the membrane and ossicles by unopposed atmospheric pressure from without. If the impairment is slight, the hearing may be practically restored to the normal, and if of more advanced grade, the improvement resulting from a first inflation is very much more pronounced than that which occurs in chronic lesions of the tympanum. This point is, therefore, an important one both in the diagnosis and prognosis of these cases.

Under chronic tubal catarrh, it may be admissible to recognize three conditions causing retraction of the drum membrane,—viz. (a) nasal

obstruction, the tube remaining patent; (b) tubal congestion; and (c) constriction due to actual hypertrophy of the tubal mucosa.

Nasal Obstruction.—There are certain cases in which, as a result of an obstructive nasal lesion, the tubes remaining open, the air in the naso-pharynx is more or less rarefied with each inspiration. In consequence of this, the air-pressure within the tympanum becomes constantly negative, and retraction of the drum membranes and some impairment of hearing result. In such a case, inflation per catheter results in a fairly good current of air to the tympanum, replacement of the drum membrane, and prompt relief of the subjective symptoms. The relief thus afforded is, however, exceedingly short lived, the patency of the tubes conducing to the quick reduction of the tympanic air-pressure under the influence of the naso-pharyngeal lesion. Obviously, in such a case, no amount of local treatment of the ears by inflations, pneumatic massage, etc., will be of any permanent value until the nasal lesion, whatever its nature, has received attention.

Chronic Tubal Congestion.—Belonging more properly to this group are those cases in which the tubal mucous membrane is the seat of chronic venous congestion. These patients present symptoms and physical signs almost identical with those just described. Inflation, however, gives unmistakable signs of obstruction due to congestion, the sounds through the diagnostic tube being at first muffled, rasping, or accompanied by moist râles, then becoming clearer as air enters the tympanum in greater volume. Inflation results in marked temporary relief of symptoms, and the gradual improvement in the auscultatory signs resulting from repeated inflations shows more or less clearly that the lesion is chiefly one of chronic venous congestion.

Another method of determining the character of the occlusion is by applying a 4 per cent. solution of cocaine by means of a bent cotton-wound applicator to the mouth and walls of the pharyngeal end of the tube.[1] Instead of cocaine, a 1 in 1000 solution of adrenalin may be used, but cocaine is preferable. If the action of the cocaine or adrenalin, thus applied, is exerted rapidly—*i.e.*, in one or two minutes—throughout the canal so as to restore its lumen to a practically normal calibre, we may assume that the obstruction is due chiefly to venous congestion. On the other hand, if inflation, following this application, does not demonstrate a notable effect upon the calibre of the canal, we may assume that the lesion is one of structural thickening, inflammatory infiltration, or at least of tissue changes more complex or advanced than simple venous engorgement. If, finally, the application of cocaine or adrenalin exerts no appreciable influence upon the calibre of a constricted tube, we must infer tentatively that the lesion is one of cicatricial or hyperplastic narrowing or stenosis. This deduction must not, however, be accepted as conclusive until we have further investigated the tubal condition by means of a

[1] The cotton applicator for the local application of drugs to the pharyngeal end of the tube is described on page 152.

Eustachian bougie. It may be that the congestion or inflammation in the pharyngeal end of the tube or in the neighborhood of the isthmus is so great as to have limited the action of the cocaine strictly to the pharyngeal end of the tube, yet this localized obstruction may yield readily to the gentle passage of the bougie. Combining these methods it is usually possible to interpret correctly the nature of the tubal lesion.

The importance of this careful preliminary investigation of the character of the tubal disease must be obvious to the reader, since it is clear that we can not determine upon a rational method of treatment until we know the character of the lesion which is to be the object of our attack.

Treatment. — Cases of tubal venous congestion usually respond readily to rational treatment. This should include correction of any condition within the nose or nasopharynx which might act as an excitant of tubal congestion; application of astringents to the inflamed pharyngeal end of the tube; occasional catheter inflation for the purpose of restoring the membrana tensa to its normal position.

For adult patients, inflation by catheter possesses among others the following advantages over the Politzer method: It enables one, by means of clearer otoscopic sounds obtained, to determine with greater accuracy the condition of the tube, and to measure the progressive improvement resulting from successive treatments. It eliminates the danger—where one ear only is diseased—of producing undue relaxation of the opposite drum membrane. With children, on the other hand, it is nearly always necessary to depend upon Politzerization.

Many of these cases require no other treatment than the use of antiseptic sprays and the local application of astringent drugs, *e.g.*, silver nitrate, argyrol, etc., to the naso-pharynx and pharyngeal ends of the Eustachian tubes.

Structural Narrowing of the Tube.—There is still another class of cases coming under the general head of tubal catarrh,—viz., cases in which the obstruction depends not merely upon venous congestion, but also to some extent upon actual increase in the normal tissue elements (hypertrophy). In more advanced cases the process may have given rise to a proliferation of new connective tissue (hyperplasia). The disease may occur as a uniform thickening of the tubal mucosa, or as localized deposits having a tendency to contract. During inflation the sounds through the otoscope are insufficient, high-pitched or may be almost whistling in character, clearly indicating the reduced calibre of the tube. With such a lesion, we are justified in grouping under the general heading of chronic tubal catarrh only those cases of very moderate impairment of hearing which show sufficient improvement after inflation to exclude serious involvement of the tympanic structures. In the great majority of cases, however, these patients do not come under the observation of the otologist until evidences of organic disease within the tympanum are also well marked. These advanced lesions of the tube will be considered in connection with the tympanic lesions to which they give rise.

CHRONIC NON-SUPPURATIVE TYMPANIC DISEASE.

In attempting to describe the two main types of chronic middle-ear catarrh,—viz., *chronic hypertrophic otitis media* and *chronic hyperplastic otitis media,* or dry catarrh,—it must be understood that they probably in most cases represent different stages of the same disease; or, in other words, that one is sequel to the other. However this may be,—*i.e.*, whether they in some cases represent the gradual conversion of a simple exudative inflammation into a hyperplastic condition in which the normal tissue elements are to a considerable extent replaced by new connective tissue, and in others originate independently as separate lesions from their incipiency,—there can be no doubt that as seen by the aurist they occur as conditions having a different pathologic basis, and also differing substantially in their response to treatment.

Chronic hypertrophic otitis media may be defined as a chronic exudative inflammation of the Eustachian tube and tympanum, whereby the lining membrane of both is greatly thickened and the calibre of the tube reduced, with resulting impairment of hearing.

Etiology.—Unfortunately, the lesion is frequently so advanced when first seen by the aurist that it is difficult or impossible to determine the original cause. Undoubtedly many cases are traceable to the presence of adenoids in early life. Among adults probably many cases originate in one or other of the following conditions, — viz., (1) recurrent attacks of subacute catarrhal otitis media, in which resolution is never quite complete; (2) frequent attacks of acute rhinitis, each attack giving rise to more or less tubotympanic congestion; and (3) obstructive nasopharyngeal lesions resulting in chronic tubal catarrh which later involves the tympanum.

The influence of gout and rheumatism is clearly a factor in some cases of chronic middle-ear catarrh, and such a diathesis should be taken into account as suggesting certain lines of treatment which may be indicated.

Pathology. — Pathologically this lesion is characterized by morbid changes within the Eustachian tube and also within the tympanum. The middle ear, as well as the Eustachian canal, is the seat of a subacute inflammation involving primarily the mucous membrane. The characteristic changes are congestion and round-cell infiltration, leading later to the formation of a variable amount of new connective tissue. As a result of these changes, the mucous membrane everywhere throughout the tubotympanic space is greatly thickened. The calibre of the tube is therefore considerably reduced, and may be practically closed to the automatic passage of air to the tympanum. Within the tympanic cavity the most pronounced changes, according to Politzer, occur in the neighborhood of the labyrinthine windows and in those situations where the ossicles are in contact with the tympanic walls. Diffuse inflammatory thickening, then, is the earliest characteristic feature of this

lesion. Naturally such changes can not fail to interfere with the mobility of the ossicles.

In addition to the diffuse changes above described, there are occasionally developed certain secondary products of inflammation, instances of which have been seen and recorded by Toynbee, von Tröltsch, Politzer, Moos, Bezold, Gradenigo, and others. These secondary changes usually take the form of adhesive bands between adjacent structures,—*e.g.*, between the crura of the stapes and the walls of the oval niche, between one or both arms of the stapes and the long arm of the incus, between the hammer handle and the promontory, or between adjacent surfaces of the ossicular joints. Contraction of these bands tends to limit still further the mobility of the ossicles. When adhesions occur between the hammer handle and promontory, the inward curvature of the membrana tensa is increased and the tensor tympani may be permanently shortened.

Symptoms.—A consideration of the varied morbid changes which may occur in the course of this lesion makes clear to us the wide variations in symptoms which different cases present. Usually the symptoms are very gradual in their development, so that the lesion may be well advanced before the patient applies for treatment. The first subjective symptom to attract his attention may be either impaired hearing or tinnitus aurium.

Impairment of Hearing.—Deafness in chronic catarrhal otitis media is usually of very gradual development and never becomes complete,—*i.e.*, unless disease of the auditory nerve or labyrinth is superadded to the tympanic lesion. If the patient comes under the aurist's care during the incipiency of the disease, the loss of hearing will be found very slight. For example, there may be no appreciable reduction of hearing power for the conversational voice, the initial loss being shown only by a very slight reduction of the hearing distance for such sounds as the watch or acoumeter. At this stage loss of acuteness for the lower musical tones and increase in bone conduction are too slight to be demonstrable. The writer has, however, frequently examined patients who could hear tones as low as 18 d. v. or 20 d. v., yet showed a beginning depreciation of one ear as compared with the other by stating that the sound was distinctly fainter in the ear involved. This of course represents the stage of the disease most favorable for a practical and permanent cure. As the lesion advances, the deafness becomes more noticeable, and the functional reactions assume a more characteristic type,—*e.g.*, the lowest musical tones are no longer heard and hearing by bone conduction is increased. The high musical tones, on the other hand, are heard with undiminished acuteness. Increase in bone conduction usually advances with the progress of the lesion. I believe, however, that Bezold was justified in his conclusion that the ratio between air conduction and bone conduction is never absolutely reversed until some structural change—*e.g.*, inflammatory adhesions, fibrous hyperplasia, or the formation of new bone (otosclerosis)—has occurred within

the oval window or niche, very markedly interfering with the mobility of the stapes. If we accept this view,—*i.e.*, that a negative Rinné means stapedial ankylosis, partial or complete,—it is clear that this test acquires added importance as throwing some light upon the character of the lesion, and therefore upon the prognosis. If the disease is unilateral or decidedly more advanced in one ear than the other, a vibrating tuning-fork held in contact with the mid-line of the skull is usually referred by the patient to the more diseased ear (Weber's test). Occasionally, however, it gives anomalous or contradictory results, presumably as a result of differences in the thickness, solidity, and conductility of the two sides of the skull. It is, therefore, of value chiefly when considered in connection with results of other tests. What is of greater concern to the patient is the progressive loss of auditory acuteness for all sounds by air conduction. Usually his first intimation of impaired hearing for the conversational voice comes when he first experiences auditory strain at the theatre or in church, or at about this time he may notice that he occasionally loses the drift of a general conversation,—*e.g.*, at a dinner party. Yet in talking with one or two persons he experiences absolutely no difficulty. From this stage, years may elapse before the deafness reaches the point where even with one or two persons conversation becomes difficult. It is, unfortunately, at this advanced stage that many patients first apply to the aurist for help.

As in tubal catarrh, but to a smaller degree, the deafness in these cases varies from time to time,—*e.g.*, with the season of the year, with weather changes, and to some extent with changes of climate. As the lesion advances and the deafness becomes more pronounced, these periods of temporary improvement become less frequent and noticeable.

Tinnitus Aurium.—Patients suffering from chronic catarrhal otitis media differ very greatly in their subjection to this troublesome symptom. To some extent it is present in nearly every case. In some cases, however, the subjective noises are so slight as not seriously to disturb the patient, and sometimes he is obliged to concentrate his mind upon them before he can affirm that they are present. In other cases the loud head noises are the patient's chief concern, and, if he be a nervous individual, they may become a veritable scourge. In nearly all cases the subjective noises vary greatly at different times, or at least the patient is more troubled by them at certain times than at others. The character of the sounds varies greatly, as described by different individuals. Usually they are high-pitched. Sometimes the patient describes two distinct sounds which are heard synchronously in the same ear. While treatment which is successful in improving the hearing frequently relieves also the subjective noises, this is not by any means invariable. It is best, therefore, to discourage the patient from concentrating his attention too closely on this illusive symptom, which may be among the last to be relieved.

Pain.—While it is true that earache is not a characteristic symptom of chronic middle-ear catarrh, I do not believe that ear pain should be

entirely omitted from the symptomatology of the disease. The writer, who has himself suffered from chronic catarrhal otitis media, has occasionally experienced pain in the ear,—sometimes starting quite sharply, but usually soon assuming the character of a dull, trying, but not unbearable earache, lasting a few hours or sometimes a day or two. I know that many sufferers have these periodic ear pains in chronic catarrhal otitis media. Many do not. In those cases in which they occur, I believe that they are in some way related to periods of active progress or exacerbation of the lesion.

Vertigo.—A comparatively rare phenomenon in chronic catarrhal otitis media is subjective vertigo. Usually it amounts to little more than slight dizziness lasting but a few moments. Apparently it is not rotary in character,—*i.e.*, it is not attended by the impression of the rotation of surrounding objects,—but is rather a momentary "light-headedness," induced by suddenly rising from a stooping or the recumbent position,—*e.g.*, after stooping to tie the shoes, on getting out of bed in the morning, etc. The exact causation is not known, the theory usually advanced, that it is caused by disturbance of intra-labyrinthine pressure brought about by inward displacement of the ossicles, being merely a hypothesis, and far from convincing.

Physical Signs.—Inspection by reflected light reveals almost invariably a retracted drum membrane, the short process appearing more than usually prominent, and the hammer handle being displaced inward and backward. In some cases the hammer handle is so far rotated inward as to appear much foreshortened, or to occupy nearly a horizontal position. In others, in which inward rotation is less marked, the hammer handle appears much broader than in the normal ear, this being due possibly to slight rotation upon the long axis, or to the folding about it of a thickened and relaxed membrana tensa. The light reflex is almost invariably changed,—being either absent, or reduced to a mere line or point, or broken up into two or more points of light. The commonest type of divided reflex is seen at a point of light in front of and below the umbo, and a second crescentic or curvilinear reflex lying parallel with the peripheral edge of the membrana tensa in the antero-inferior quadrant. When retraction is marked, the posterior fold and the annulus tendinosus appear unusually prominent. The physical signs of retraction are practically the same as are seen in acute tubal catarrh (Figs. 94, 95, and 96).

In some cases the earliest stages of the disease are attended by an effusion of serum into the tympanic cavity. This fluid rarely or never fills the cavity, but may rise well above the level of the floor of the bony canal. A condition described in many text-books, but rarely seen, is that of a hair-like line running more or less horizontally across the membrana tensa, representing the upper surface level of this fluid. Naturally such a line would change its direction with changes in the position of the head.

Other than the physical signs of retraction, the drum membrane may

present no visible evidences of disease. On the other hand, there may be certain abnormalities due to structural changes.

Calcareous Thickening.—The drum membrane may present the appearance of being thickened within certain well-defined areas as if by a chalky deposit upon its inner surface or within its structure. These localized deposits are often crescentic in shape and confined to the posterior segment (Fig. 138), but may take any form and occupy any part of the membrana tensa. They are commonly regarded as calcareous deposits resulting from a rheumatic diathesis. Apparently they do not *per se* cause much disturbance of function, since they are frequently seen in persons of normal hearing.

Fig. 138.—Thickening due to calcareous deposits.

Tubal Conditions.—During catheter inflation the sounds through the diagnostic tube may be faint, indistinct, and distant,—indicating an obstructed tube through which little or no air enters the tympanic cavity. This, after several compressions of the bulb, may be succeeded by a blowing sound, giving the impression of sound originating in or near our own ear, indicating that the tube has suddenly opened under the pressure of air from the inflating apparatus. This sudden and very marked improvement in the character of the sound while inflation is in progress usually means that congestion plays a prominent part in the tubal lesion.

In other cases air may be heard entering the tympanum from the start, the sounds being unmistakably tympanic in character, but accompanied by moist râles. Such sounds usually indicate an exudative inflammation of the tube, accompanied by an increased secretion of mucus, which partly occludes its lumen. Not infrequently such râles disappear or become fainter and less frequent during inflation, as a result of displacement of the tubal secretion which is expelled into the pharynx by the return air current.

Structural narrowing of the tube is characterized by a sound of tympanic quality, but fainter and of higher pitch than is normally heard,—fainter by reason of the small volume of air entering the tympanum, the higher pitch being explained by the reduced calibre of the tube. Absolute closure of the tube, which is exceedingly rare, would be indicated by complete absence of sound characteristic of air entering the tympanum.

Obviously no trustworthy deductions can be drawn from catheter inflation until the student has acquired sufficient technical skill to feel confident of his ability to place the tip of the catheter in its correct position in the pharyngeal end of the tube.

Besides determining the tubal condition, the otoscope also throws some light upon the tympanic lesion, particularly as to the presence of two conditions occasionally resulting from this lesion,—viz., the presence of fluid and relaxation of the drum membrane.

In some cases of chronic hypertrophic otitis media, a limited amount of mucoserous fluid collects in the lower part of the tympanic cavity. Usually it is not sufficient in amount to influence the position of the drum membrane, and may be quite indeterminable by inspection. In such a case the inflation sound is modified by a series of bubbling râles. The sound is so distinctly characteristic of the passage of air through fluid as to be quite unmistakable. As a comparatively rare result of inflation in these cases, a number of bubbles may be blown up upon the inner surface of the membrana tensa, which then become visible through the membrane by reflected light.

Relaxation of the drum membrane is demonstrated during inflation when each otoscopic sound begins with a distinct snap. Inflation of the normal ear does not give rise to a percussion sound due to the impact of air upon the drum membrane; it begins and ends as a moderately low blowing sound. With moderate relaxation of the drum-head, there is heard with each inflation a short snap, immediately followed by the characteristic "blow." With extreme relaxation, there are two distinct and characteristic flapping sounds,—one at the beginning of inflation as the relaxed membrane is blown outward, and a second at the end, due to its recoil. In this way we frequently become aware of marked changes in tension which may be absolutely indeterminable by inspection alone.

This loss of normal tension, or relaxation, may be further corroborated by means of Siegel's otoscope.[2] Through the speculum of this instrument the relaxed membrane is seen to execute exaggerated movements,—inward against the inner tympanic wall and outward into the auditory canal,—as the air in the meatus is alternately compressed and rarefied. At the same time we may note a characteristic of the relaxed drum membrane which accounts in large part for the resulting deafness,—viz., the relatively small influence which wide movements of the relaxed membrane exert upon the hammer handle, and therefore upon the ossicular chain.

The hearing in chronic hypertrophic otitis media is in nearly all cases improved as an immediate result of a first inflation. In the early stages this improvement is especially marked. As the disease advances and the pathologic changes within the tympanum involve more seriously the ossicular joints, the functional gain following inflation is naturally less pronounced.

Chronic hypertrophic otitis media tends to gradual but progressive impairment of hearing. Whether the functional loss finally disables the patient for enjoying the society of his fellows depends to some extent upon whether he is fortunate enough to come early under proper care, and also upon the character of the lesion. It is conceivable that two cases developing with the same initial severity may within the same period of

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[2] See Fig. 59, page 59.

years reach very different grades of deafness, from the fact that the hypertrophic process may in one case involve chiefly the regions of the ossicular joints, and in the other may be spread over regions of less functional importance. Fortunately, the disease may enjoy periods of apparent quiescence, and, even when this can not be affirmed, the patient may be well past middle age before his deafness becomes a serious handicap in life. There is, however, a numerous class of patients with whom impairment of hearing begins very early in life and progresses rapidly, so that the individual is seriously hampered at a time when his career is still in the formative stage and his best energies still called for.

Prognosis.—In the very early stages the disease is probably a curable one. In its later stages, when the pathologic basis of the symptoms is found in gross morbid changes which can no longer be corrected, the patient's symptoms may be relieved and his hearing for a time improved, but the lesion is no longer curable.

Treatment.—It is clear that this lesion may give rise to conditions differing widely in their mechanical influence upon sound-conduction. Thus, in one case deafness may be due very largely to tubal obstruction causing retraction of a relatively normal drum membrane, while in another the tube may be patent and the deafness due chiefly to abnormal relaxation of the drum-head. In still a third variety neither tubal obstruction nor relaxation of the drum-head may be marked, the deafness depending chiefly upon adhesive processes involving the oval window and stapes. Obviously successful treatment demands recognition of the mechanical cause of deafness.

Care of the Nose and Nasopharynx.—As a preliminary measure, the nose and nasopharynx should be carefully examined and any lesion or condition interfering with nasal respiration, or acting as a predisposing cause of tubal congestion, should be corrected. Preliminary to the use of the catheter, the nose and nasopharynx should be sprayed with some alkaline cleansing solution (Dobell's, alkalol, normal salt solution). This periodic flushing of the nasal spaces is in itself distinctly beneficial in some cases. When the turbinates and the nasal mucosa are in a state of chronic congestion or turgescence, the occasional application of argyrol is often of value. Under its influence, the congestion usually subsides and the mucous membrane regains a more normal tone and appearance. Argyrol may be applied twice or three times a week,—either locally in 25 per cent. solution by means of a cotton applicator, or in 5 per cent. solution in the form of a spray.

The home use by the patient of cleansing sprays followed by oil sprays leads to improvement in some cases, in others seeming rather to excite or perpetuate a subacute form of nasal congestion or irritation. Their results should, therefore, be carefully watched.

Local Treatment.—The Eustachian tube is probably in most cases the starting-point of this lesion and is our logical point of attack. If the condition here is chiefly one of venous congestion, it will in most cases

respond to the measures outlined under chronic tubal congestion, which need not be repeated in detail here.

In all cases in which the tubes are partially occluded, the local treatment should begin with inflation, preferably by catheter. Inflation should be repeated at regular intervals, at first on alternate days, this being continued as long as progressive improvement in hearing can be demonstrated. By occasional retesting, the results being compared with those of the original functional examination, it will be easy to gauge the results of treatment. When the functional gain reaches a point beyond which no improvement can be demonstrated, the inflations should be stopped, or practised only at considerably longer intervals.

In many cases the disturbance of the tubal function is due largely to persistent inflammatory changes in the pharyngeal end of the tube, in which case the local use of astringent drugs forms an essential part of the treatment. They are applied by means of a cotton applicator bent to a curve similar to that of the Eustachian catheter, and introduced catheter-fashion into the mouth of the tube. In my experience, nitrate of silver in solution of gr. x-xxx ad ℥j, and argyrol in 25 per cent. solution, are the drugs which give the best average results. While nitrate of silver is usually well tolerated by the tubal mucosa, it is best to begin with a weak solution,—*e.g.*, gr. x ad ℥j, —and later increase the strength to gr. xxx to the ounce, which accomplishes all that would result from a stronger solution and is less irritating. This drug should not, as a rule, be applied to the tube oftener than once in seven days. It causes considerable burning discomfort at the time, which, however, usually subsides within from thirty minutes to an hour, leaving the tubes clearer. Its value may be considered proved in those cases in which the beneficial results of inflation are thereby prolonged or rendered more stable. There are cases, however, in which nitrate of silver increases tubal congestion and is distinctly harmful. I have personally found argyrol of more universal value than nitrate of silver. It is quite as astringent as the latter, has very decided antiseptic value, and, so far as I have been able to observe, is practically non-irritating. It may be used in solutions from 25 to 50 per cent., and as often as twice or three times a week. Next to nitrate of silver and argyrol, I have found chloride of zinc in 2 to 4 per cent. solutions of most value in these cases.

Should inflation and the local use of astringents fail to restore the functional activity of the canal, the Eustachian bougie must be employed. In the author's opinion, there are but three clearly defined indications for the use of the Eustachian bougies: (1) to determine and locate a point of local narrowing, or constriction; (2) to overcome a constriction due to inflammatory adhesions within the membrano-cartilaginous portion of the tube; and (3) in certain cases of obstinate tubal congestion, in which the passage without force of a small bougie helps to re-establish the tubal function and opens the way for the local or direct application of astringent drugs. Other than the above definite and distinct indications, I believe that the frequent use of bougies is likely to do more harm than good.

Eustachian bougies are made of whalebone, celluloid, or certain com-

positions in which gum elastic is a prominent constituent. They all terminate in an olive-shaped enlargement or bulb. This terminal enlargement is of diagnostic value in determining the exact position of any localized constriction, in that the stricture offers resistance to its passage inward, and again to its withdrawal. Thus, a bougie may pass easily through the tube until it meets resistance from a localized constriction. Passing this, it may enter the tympanum without further opposition or it may encounter and pass a second stricture. Owing to its terminal bulb, its withdrawal is again opposed at the points of resistance previously noted. In this way one or more strictures may be determined. The size is measured by the diameter of the terminal bulb, and varies from a half millimetre or less to one and a half millimetres, the latter measurement being the largest that should be used. Of the different kinds of bougies, the celluloid are much more pliable than the whalebone, following the curve of the tube better, and being therefore less likely to create a false passage. Most of the composition bougies are open to the objection that they soon become rough, increasing the danger of injury to the tubal mucosa.

As to the size of bougie to be used, whoever attempts this rather delicate procedure should remember that the average normal tube at the

Fig. 139.—Celluloid bougies (calibre and length reduced).

isthmus is one and a half millimetres in diameter; some are smaller. If we employ a bougie which is too large for the tube, we may by the application of force carry it beyond the isthmus, but in doing so may crush or cut the mucous lining against the beginning of the bony tube, giving rise to an annular cicatrix whose subsequent contraction may cause the most intractable kind of stricture. Ordinarily a bougie with a bulb one millimetre in diameter should first be tried. If it meets too much resistance at the isthmus, it is better that it be withdrawn and a smaller one substituted.

Technic.—As a preparatory step, we will assume that the nose and nasopharynx have been thoroughly sprayed with Dobell's or other cleansing solution; that the catheter of malleable silver has been sterilized by boiling; that the celluloid bougie, which cannot be boiled, has been cleansed in alcohol and dried with sterile gauze.

We must also assume that the physician has clearly in mind the following facts, without which he cannot work intelligently: 1, that the Eustachian tube in adults varies from 31 millimetres to 38 millimetres in length; 2, that its narrowest part, the isthmus, is at the junction of the bony with the membrano-cartilaginous portion of the tube; and 3, that the end bulb of the bougie will reach the isthmus when it has passed beyond the terminal opening of the catheter to a distance varying between 20 and 25 millimetres.

Obviously we must adopt some method by which the physician may know at any time just how far the bougie has passed out of the catheter and into the Eustachian canal. In practice I meet this necessity in the

following way. Taking, for example, a celluloid bougie, I pass it through the catheter end to a length of about 38 millimetres, or about one and a half inches (Fig. 140). Practice soon enables one to gauge this measurement quite accurately by eye. I now bend the opposite, or proximal, end quite sharply downward over the edge of the large end of the catheter. The bougie retains this bend or angle (*b*). Now, when the catheter is

Fig. 140.—Eustachian catheter and bougie.

in proper position in the patient's nose and pharynx and I have introduced the bougie, I know, when my guide-bend is 38 millimetres from the outer extremity of the catheter, that the tip of the bougie is just entering the canal. As the bougie is advanced into the canal, I am able at any time to compute the distance it has traversed by mentally deducting from 38 millimetres the distance remaining between the expanded extremity of the catheter and the bend in the bougie.

The bougie should be passed very slowly and with little application of force. If one uses a very small bougie, it may traverse the whole length of the canal with no appreciable resistance. With a terminal bulb one millimetre in diameter, one will almost invariably meet with a sufficient increase in resistance at the isthmus to inform one that the bougie has reached this narrowest point of the canal. If the isthmus can not be passed without the use of considerable force, it is better to withdraw the bougie and either employ a smaller one, or try the same bougie at a later sitting after very thoroughly shrinking the canal with cocaine or adrenalin. In many cases the passage of a bougie of proper size is followed within a few days by a very noticeable improvement in the patency of the tube.

Yankauer[3] has devised a set of Eustachian applicators, bougies, and sounds, and described in detail a systematic method of dilating a constricted tube, which, while founded upon methods long in use, represents a useful technical advance. By this method it is possible not only to locate, but also to determine the extent, measured in millimetres, of the constricted portion of the tube. This constricted portion is then anæsthetized and exsanguinated by a 5 per cent. solution of cocaine in adrenalin, 1 to 2000, carried into the tube by a specially devised cotton applicator. Following this, inflation demonstrates whether the constriction is due to congestion or to inflammatory infiltration of the tubal mucosa. If due to the former,

[3] Yankauer: The Isthmus of the Eustachian Tube, A Contribution to the Pathology and Treatment of Middle-ear Disease, Laryngoscope, July, 1910.

application of 25 per cent. or 50 per cent. argyrol solution is made to the diseased portion. If dependent upon inflammatory infiltration, the application of argyrol is preceded by the passage of the Eustachian sound, which is allowed to remain in place five or ten minutes. While this method represents a useful initial measure in certain cases, its frequent repetition as advised by Yankauer, does not impress the writer as advisable, or as altogether without risks.

The handles of Yankauer's instruments are provided with a graduated scale, enabling the physician to tell at any moment the exact distance to which the bougie or sound has entered the canal.

The Electric Bougie (*Duel*).—Still another method of dilating an obstinate stricture of the tube is by the so-called "electrolytic method" devised by Dr. A. B. Duel, of New York. Basing his experiments upon the hypothesis,—which is an accepted fact in electrotherapeutics,—that contact of the negative pole of a galvanic current favors absorption of inflammatory products, he subjected a number of patients, with tubal strictures which had resisted other methods, to the following treatment: Using a galvanic battery provided with a reliable rheostat and milliamperemeter, a bougie constructed of gold wire was connected with the negative pole, the positive electrode being held in the patient's hand. This bougie was passed through a silver catheter, insulated by a covering of rubber tissue, and slowly advanced until its progress was blocked by the stricture. The current was then gradually turned on to a strength varying between two and five milliamperes. In many cases it was found that at the end of from two to five minutes the bougie could be advanced without force.

A somewhat alarming accident, which has been recorded by several aurists, is the breaking or separation of the distal end of the bougie within the Eustachian tube. This has never occurred in the writer's experience, and he has found this treatment of decided value in certain cases in which all other methods of dilatation had failed. It is an agent which should not be used carelessly, and whoever wishes to employ it should follow carefully the technic as described by its sponsor.[4]

The patency of the tube having been restored so far as catheter inflation may indicate, the patient should for a time be carefully observed for the purpose of determining whether the tubal function has also been re-established. Every aurist has had experience with cases in which inflation results in an apparently normal air current to the tympanum, yet with each return of the patient the drum membrane is found retracted. Functional improvement following inflation is, therefore, only temporary. This condition would seem fairly positive proof that, while the tube itself is patent, its function has not been resumed. Upon such a case a comparison of the results of inflation by catheter and by the Politzer method may throw considerable light. Supposing, for example, in a case of unilateral disease that catheter inflation reveals a normally patent tube and

[4] Duel: The Technic of Eustachian Electrolysis, Trans. Am. Lar., Rhinol., and Otol. Society, 1909.

yet by the Politzer method little or no air reaches the tympanum, the evidence of obstruction at the pharyngeal mouth of the tube would seem to be clear. The mechanical cause of this obstruction may or may not be visible by means of the postnasal mirror, being due possibly to one or other of the following causes,—viz.: (a) remnants of adenoid tissue remaining in the fossa of Rosenmüller, occasional congestion of which closes the tube by pressure; (b) chronic œdema or swelling of the lips of the pharyngeal mouth of the tube, giving them a valvular action under pressure; (c) unrelieved œdema within the mouth of the tube, beyond which the catheter passes; and, lastly, (d) there may possibly be cases in which, as a result of hyperplastic thickening or induration of the nasopharyngeal and tubal tissues, the pharyngeal muscles no longer act effectively upon the pharyngeal end of the tube. These conditions suggest their appropriate treatment.

When finally the function of the Eustachian canal has been re-established, the further treatment will naturally depend upon the interpretation of the tympanic condition. If the subjective symptoms have been relieved and a reasonable improvement of hearing obtained, it may be better to suspend all treatment, instructing the patient to return within three to six months in order that the permanency of the results may be tested. On the other hand, if symptoms are present depending apparently upon tympanic hypertrophy or congestion, a persistent effort should be made to correct these conditions.

Since any further treatment which may be required in these cases conforms in a general way to that indicated in the hyperplastic form of chronic otitis media, the subject may with advantage be deferred to be considered jointly with the treatment of that disease.

Chronic hyperplastic otitis media (sometimes called dry catarrh of the middle ear) is characterized by an excessive production of new connective tissue within the tympanic mucosa. In the drum membrane this hyperplasia may involve not only the mucous membrane, but also the fibrous layers of the membrana tensa, which may to a greater or less degree be replaced by new connective tissue. The mechanical results of these changes vary logically with the regions and tympanic structures involved. Wherever the joint structures of the ossicular chain are involved, the contraction of new connective tissue may react unfavorably upon ossicular mobility. With the membrana tensa, on the other hand, while connective tissue hyperplasia may lead to apparent thickening, the loss or atrophy of the normal fibrous layers may, and usually does, result in a loss of normal tonicity, or relaxation, which constitutes one of the chief factors in the mechanics of obstructive deafness.

All authors agree that the condition above described is often a late stage of the hypertrophic form of chronic middle-ear catarrh, beginning with well-marked round-celled infiltration, and ending in the conversion of the newly formed round cells into fibrous connective tissue. The same process, taking place within the Eustachian tube, may give rise to widely different end-results,—*e.g.*, (a) the deposition of new tissue may be so extensive as

nearly to occlude the isthmus of the tube, resulting in an organized obstruction or stenosis very difficult to correct; or (b) the hyperplastic process being less marked, contraction of a moderate amount of new connective tissue gives rise to an abnormally wide and open tube.

The lesion may be further complicated by the presence of fibrous bands passing between the ossicles and adjacent tympanic surfaces. They may, for example, bind the head or crura of the stapes to the walls of the oval niche, adding greatly to the impairment of hearing. Such adhesive structures probably always represent an earlier hypertrophic or exudative stage in which swelling or œdema was a more or less prominent feature.

SYMPTOMS. — Since they in many respects resemble those already described under chronic hypertrophic otitis media, the symptoms may be dealt with briefly.

Usually when the diagnosis is made the hearing is already considerably impaired, there being noticeable loss of hearing for the lowest musical tones, and hearing by bone conduction being appreciably prolonged. The ratio between hearing by air conduction and bone conduction may or may not be reversed, actual reversal not taking place until the deafness is very pronounced. As compared with chronic hypertrophic otitis media, it has seemed to me that sufferers from this lesion show often a loss of hearing for the conversational voice and whisper out of proportion to the demonstrable changes in tone limits and bone conduction. For example, the patient may have difficulty in hearing a moderately loud whisper at 5 or 6 feet, and yet may hear forks as low as 36 or 40 double vibrations per second, and bone conduction may be only moderately increased. The degree of deafness is also less variable, neither weather conditions nor changes of season or climate influencing it so markedly as in the hypertrophic form of middle-ear catarrh.

Paracusis (*Willis*).—A condition which sometimes accompanies the deafness of this lesion is known as paracusis Willisii. It is present when an individual with advanced deafness can understand conversational speech better in the presence of loud surrounding noises than in a quiet room. Thus, some patients, who are ordinarily extremely deaf, hear with comparative ease while traveling in a railway-car or when walking in a noisy street. There are two theories in accordance with which this phenomenon is explained,—viz.: (1) The tympanic theory,—*i.e.*, that, with pronounced tympanic changes, the ossicular chain loses the power of responding adequately to the conversational voice; but that, when set in motion by such gross sounds as the noises of the street or of a railway-car in motion, they regain for the time their responsiveness to the more delicate sound waves involved in conversational speech. (2) The labyrinthine theory is that in cases of very advanced deafness, even though the lesion may be of tympanic origin, the auditory nerve becomes more or less torpid and irresponsive to the voice sounds, but that when stimulated by loud noises it becomes simultaneously more responsive to the more delicate and complex sound waves of the conversational voice.

Paracusis is seldom present in chronic hyperplastic otitis media except in the advanced stages in which the ratio between air conduction and bone conduction is reversed (negative Rinné). It is also very commonly present in advanced stages of otosclerosis. It is probably, therefore, in some way related to conditions bringing about fixation of the stapes, and to this extent is of some importance as bearing upon the prognosis.

Tinnitus aurium, though usually present, varies greatly in different cases, either in its intensity or in its impression upon the patient. Some patients, while stating that the subjective noises are continually present, do not seem to be greatly disturbed thereby. Others, either by reason of their loud character or through anxiety as to their significance, are made exceedingly nervous or unhappy by them. A not uncommon impression among those who suffer from tinnitus is that their difficulty in hearing is due chiefly to the confusion incident to the subjective noises, rather than to actual impairment. "I could hear perfectly if it were not for these head noises," is a statement with which every aurist is familiar. Fortunately, many people with incurable ear lesions become so accustomed to continual head noises as actually to be unconscious of them during the day, when the noises of the street and the interests of the hour serve to distract their attention. Undoubtedly individuals of trained will power are less disturbed than the weak, nervous, or neurasthenic.

As with chronic hypertrophic otitis media, vertigo is a comparatively rare phenomenon, and when it does occur is usually little more than a momentary dizziness, sometimes, however, recurring with annoying persistence. When severe attacks of vertigo, accompanied by disturbance of static equilibrium, occur in this lesion, careful functional examination will usually establish other clearly defined evidences of labyrinthine involvement.

Physical Appearances of the Drum Membrane.—While the Eustachian canal may be not only patent but abnormally wide, it is a curious fact that the drum membrane is in most cases distinctly retracted. The frequent coincidence of a widely open tube and a retracted membrane may in some cases be accounted for as being the result of adhesions between the hammer handle and promontory, shortening of the tensor tympani, etc. Probably much oftener it is the result of an earlier hypertrophic stage, during which tubal obstruction led to prolonged retraction of the membrane, the resulting loss of resiliency preventing its subsequent return to the normal position.

The most characteristic appearance, however, is that of moderate thickening,—the membrana tensa appearing to be not only thicker but to have lost in some degree its characteristic sheen or lustre. This condition is usually the result of an increase of connective tissue at the expense of the normal fibrous layers of the membrana tensa. The apparent thickening is often most marked at the periphery and in the central area immediately surrounding the umbo. In some cases the deposition of new connective tissue may be plainly seen in the form of opaque lines running

in various directions through the tense membrane, most often radiating from the umbo or hammer handle to the periphery. Such a drum membrane, if examined with Siegel's otoscope, will not infrequently be found to be abnormally relaxed, both anterior and posterior segments making exaggerated movements as the air in the auditory canal is alternately compressed and rarefied.

Atrophy of the Drum Membrane.—The drum membrane may present exactly the opposite appearance to that just described, the whole membrana tensa being abnormally thin and transparent. This change is probably the result of atrophy and partial disappearance of its fibrous layers under the pressure of a moderate deposition of new connective tissue. The membrane in consequence is exceedingly thin, transparent, and so relaxed as to exhibit in some cases exaggerated degrees of retraction. Tympanic structures not clearly visible through the normal membrane come therefore plainly into view through the depressed and attenuated membrane. Thus, the incudostapedial joint and niche of the round window may be quite prominent, the membrane being applied so closely to the inner tympanic wall as almost to give the impression that one is looking directly at the tympanic structures. This is the type of membrane which may be easily ruptured by catheter inflation even with moderate pressure. Naturally the physical picture is greatly altered by inflation, but the lack of normal tension favors quick return to the condition of exaggerated retraction.

There are also cases in which the membrana tensa undergoes atrophy only in certain regions, the rest of the membrane being apparently normal. This condition is in some cases easily recognized by the greater transparency of the area involved; in others, there may be no structural change which the eye can appreciate, the condition being detected only while examining the drum membrane with a Siegel otoscope, when the atrophied part is seen to balloon out into the meatus independently of the surrounding normal membrane.

The immediate effect of inflation upon the hearing depends upon a variety of conditions,—*e.g.*, patency or structural narrowing of the tube; the degree of relaxation of the membrana tensa; and, more than any other condition, upon the presence of adhesive processes involving the stapes and margins of the oval niche. Whatever the condition may be, the immediate effect of a first inflation is usually in the direction of an improvement in hearing. As a rule, however, the functional gain is not so pronounced as that which is obtained in chronic hypertrophic otitis media. This is to some extent a differential point between the two conditions. When the tube is greatly narrowed at the isthmus, the otoscopic sound during inflation is of reduced volume and higher pitch than is normally heard. On the other hand, with an abnormally wide tube, one hears a very low, full, and often rough, blowing sound. A very common accompaniment of the pathologically wide tube is marked relaxation of the drum membrane. During inflation one may hear, therefore, not only the low, rough, blowing

sound of the wide and probably dry canal, but in addition the characteristic flapping sound of the relaxed membrane. In such a case the increased calibre of the tube and loss of tension in the membrana tensa are each in their way expressions of the same morbid process,—viz., atrophy and partial disappearance of normal tissue elements.

The use of Siegel's otoscope is an important part of the examination of these cases, enabling the physician to detect adhesions between the hammer handle and promontory, or between the drum membrane and intratympanic structures. During rarefaction of air in the meatus, the free portions of the drum membrane are sucked outward into the canal, leaving the points of adhesion depressed and in conspicuous view. The Siegel otoscope also enables one to determine loss of tension—*i.e.*, relaxation—of the membrane, either localized or involving the whole membrana tensa.

Course of the Disease; Prognosis.—The tendency of the disease is toward slowly increasing deafness. The possibilities of treatment depend in each case upon the stage of the disease, the degree of deafness, and the physician's ability to interpret correctly the mechanical cause of the deafness. In some cases local treatment fails utterly to influence the symptoms. In most cases, however, something may be accomplished either in the way of improving the patient's hearing or conserving that which remains to him.

Treatment.—Since the treatment of chronic hypertrophic otitis media has been described somewhat in detail, it will suffice here to speak briefly of the chief indications in this form of chronic middle-ear disease.

As with all forms of tympanic disease, the Eustachian canal should receive first attention. If the tube is found to be structurally narrowed, an effort should be made to re-establish its normal patency. For this purpose some form of mechanical dilatation is essential. Preparatory to the introduction of a bougie, the nose should be thoroughly sprayed with some cleansing solution (Dobell's, Seiler's, alkalol), and a 4 per cent. solution of cocaine should be applied to the pharyngeal end of the tube. Using the ordinary celluloid or whalebone bougie, it is best to employ first one of the smaller sizes, gradually advancing to the largest size which will pass the isthmus without much force.

A weak point in this type of dilator is in the form of the bougie,—*i.e.*, a terminal enlargement or bulb attached to a narrow shaft,—limiting the dilation to the passage back and forth of the bulb. To meet this difficulty Yankauer has devised a Eustachian sound, the tubal part of which is of uniform calibre, enabling one to maintain uniform dilatation throughout the constricted portion of the tube. This in some cases gives better results than the ordinary bougie.

Whatever instrument is used, its calibre should not be larger than can be made to pass the isthmus with comparative ease,—*i.e.*, without risk of injury to the tympanic mucosa. The effort by suddenly applied force to pass a large bougie beyond the isthmus may give rise to traumatism

and subsequent cicatrization, leading ultimately to an annular constriction, or stricture, far more difficult of correction than that which we set out to relieve.

Following the sound or bougie, the application of a 25 per cent. solution of argyrol throughout the entire length of the canal by means of a Eustachian applicator may add to the efficiency of this treatment.

The use of bougies has been advocated by various authorities and for various purposes. It has been used, for example, as a means of massaging the tubal mucosa, for which purpose a bougie as large as will pass the isthmus without force is introduced, and allowed to remain in the tube five minutes or more. Professor Urbantschitsch advised that its stimulating action be increased by friction, the bougie being moved rapidly back and forth in the dilated tube, a measure which would seem to the author to be

FIG. 141.—Eustachian applicator made of No. 5 piano wire

contraindicated by the inherent risks. With the same end in view, the tubal end of the bougie may be dipped into a solution of arygrol or ichthyol or into a weak solution of nitrate of silver, and passed thus into the canal. Albert A. Gray, of Glasgow, advises that the end of the bougie be smeared with an ointment having a one-half per cent. nitrate of silver content, the vehicle being lanolin. The bougie thus medicated he allows to remain in the tube twenty minutes or longer. All of these measures have in view the purpose of increasing the local blood supply of a wide-open and sclerotic canal.

Probably the most effective mode of stimulating the tubal membrane, and certainly the best way of applying drugs locally to the canal, is by means of a suitable Eustachian applicator. A perfectly practical applicator and one which can be easily prepared by the physician, is that long since described by Dr. Dench. It consists simply of a piece of No. 5 piano wire cut to the proper length, the end of which to a length of about one millimetre is sharply bent backward so as to lie in contact with the main wire (Fig. 141).

A very small pledget of sterile cotton wound about this is firmly engaged by the terminal hook, and cannot be dislodged in the tube. Yankauer's applicator has the advantage of a convenient handle and greater flexibility (Fig. 142). With either of these instruments drugs may be applied throughout the entire length of a patent tube. In the author's experience, argyrol in 25

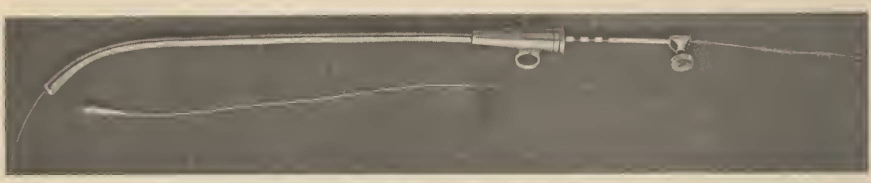

Fig. 142.—Yankauer's Eustachian applicator.

to 50 per cent. solution is the drug giving best results in these cases; it is both astringent and stimulating, possesses considerable antiseptic value, and is practically non-irritating. It may be applied as often as twice or three times a week.

After all that seems possible in re-establishing normal conditions within the Eustachian canal has been accomplished, there is still the question of the tympanic cavity proper, and what may be done to restore its lining membrane to a condition more nearly approaching the normal standard.

Upon purely theoretic grounds it would seem that drugs which have proved their value as applied to the tube should have a similar field of usefulness as applied to the tympanic cavity. That they are not equally available is due not to any difference in their local action in the two situations, but to the comparative difficulty in applying them to the tympanic walls. While the introduction into the tympanum of various drugs in solution has been mentioned by many distinguished aurists, I have always felt that this might be attended with certain risks. My own experience has, therefore, been confined largely to the use of one drug, argyrol, applying it in the following way,—viz., the Eustachian canal having been cocainized, a few drops of a 25 per cent. solution of argyrol are taken up in the end of a catheter, which is then introduced and inflation performed in the usual way. In this way a drop or two of the solution reaches the tympanum, probably in the form of a spray. If the drum membrane is inspected a minute or two thereafter, it will be found to be markedly congested. The patient usually experiences a burning sensation, but rarely pain. Both the sensation of heat and the redness are temporary, and the congestion will usually have disappeared when the patient is again seen by the physician. Certain cases have been benefited by argyrol used in this way.

Catheter Inflation.—According to my experience the value of catheter inflation in the treatment of deafness is distinctly limited. The prolonged routine use of catheter, repeated at short intervals, has practically no value in correcting ossicular rigidity, and is likely to produce structural changes in drum membrane leading ultimately to definite diminution of hearing.

While in a general way it may be stated that most cases of chronic hyperplastic otitis media are benefited by occasional catheter inflation, this statement calls for an accompanying word of caution. Inflation, like every other therapeutic measure, has its indications and its contra-indications, and there can be no doubt that many cases of "chronic deafness" have been made permanently worse by irrational, routine use of the catheter. For example, let us consider a case of extreme atrophy of the drum membrane in which the membrana tensa is attenuated, transparent, and appears as if plastered against the promontory. Such a drum membrane may be easily ruptured by catheter inflation, and, if this accident does not occur, the already pathologically relaxed membrane may be still further stretched. I have seen cases presenting this extreme degree of attenuation and retraction in which a very fair amount of hearing power was retained, the drum membrane of course having ceased to fulfil any other purpose than that of a protective covering, and the sound waves being taken up directly by the ossicles beneath,—possibly being transmitted directly to the incudostapedial articulation. Under these conditions, it may be well at very considerable intervals—certainly not oftener than once or twice a month—to practise very gentle inflation simply to lift the membrane away from its contact with tympanic structures and thus prevent adhesions which, becoming organized, might add seriously and permanently to the impairment of hearing.

Another condition more easily overlooked is that in which the drum membrane, though thickened by connective-tissue hyperplasia, has suffered a coincident loss of tension,—*i.e.*, is relaxed. In this condition it is clear that catheter inflation is an exceedingly dangerous procedure, since its mechanical results are more in the direction of a further reduction of tension than of effectively exercising the ossicular chain.

If, as sometimes occurs, some degree of relaxation of the drum-head coexists with tubal or other conditions calling for catheter inflation, it may be well to apply a coating of collodion to the membrana tensa. This in contracting tends to draw the drum membrane outward into its normal position, and to protect it from further stretching under gentle inflation.

Auditory Massage.—A somewhat perplexing problem in the treatment of these cases is the necessity of combating tympanic and ossicular rigidity by some form of passive exercise. The method which has been most extensively employed for this purpose is what is known as pneumatic massage.

Pneumatic massage is a term applied to passive movements of the drum membrane by means of any instrument producing alternate condensation and rarefaction of the air in the external auditory meatus. A very large number of such instruments have at various times been placed on the market. They are all somewhat similar in design, in that they combine the principle of a Siegel otoscope with that of an electric or other motor by which the piston is moved rapidly back and forth.

Such instruments seem to be based upon the theory that whatever

will move the drum membrane and ossicles will improve the hearing. They quite ignore the ease with which the adjustment of so finely balanced a mechanism may be disturbed. Let us recall for a moment the character of this mechanism,—*i.e.*, a system of delicate and sensitively mobile levers capable of responding to the slightest movements of the drum membrane, and designed to respond to such movements, and to no others, through life. Obviously the perfect execution of this function must call for a definite and proportionate degree of tension in both drum membrane and ossicular chain, that of the latter being maintained by the tympanic muscles. Clearly the movements of a relaxed membrane can be but imperfectly transmitted to a system of heavier bone levers. If, therefore, we close the meatus and subject the drum-head to a series of crude exercises in which the ossicles can not possibly take part, it is obvious that the net result will be undue relaxation of the membrana tensa and further impairment of hearing.

In any system of tympanic massage we should bear in mind the very limited maximum excursions which, according to the investigations of Helmholtz, Politzer, and others, the individual ossicles can make in response even to experimental movements of the drum-head,—*i.e.*, hammer handle, three-fourths of a millimetre, stapes one-fourteenth of a millimetre.

In some cases a very noticeable gain in hearing has resulted from a first treatment by this method. This initial gain has rarely proved permanent, and progressive improvement has never to my knowledge resulted from repeated use of this measure.

The unsatisfactory results of this form of massage are probably explained by two facts: (1) The movements of the membrana tympani are not those which nature requires of it or which are necessary to the transmission of sound waves, and do not therefore exercise the drum membrane and ossicles in their normal function. (2) The undue strain to which the drum membrane is subjected tends to produce alterations in tension, reducing still further the patient's hearing power.

It may seem from the above that my purpose is to discourage all efforts to exercise the drum membrane and ossicles through the external auditory meatus. This, however, is not my intention, since I believe this to be one of the most important problems before otologists to-day.

Preobraschensky, of Moscow, who has made systematic investigations of the various methods in use, records his belief that vibratory massage with any instrument which fits into and tightly closes the meatus, produces such rough and exaggerated movements as to constitute an insult to the delicate mechanism involved. With this view I have long been in accord.[5]

Undoubtedly there are many cases which call for some form of ossicular massage beyond that which catheter inflation affords. In trying to meet this indication, we should bear in mind the following points: The ear-

[5] Kerrison: Notes on Some Very Simple Experiments as to the Influence of Sound Waves on Ossicular Rigidity, Jour. Amer. Med. Assoc., December 1, 1906.

piece of the instrument should not be introduced into the meatus so as to close the canal. Either it should be held slightly separated from the ear, or the ear-piece should be perforated. Tight closure of the canal produces exaggerated movements of the drum-head which are distinctly injurious. Since the loss of mobility is usually greatest in response to the relatively slow vibrations concerned in the production of low musical tones, it would seem more rational to employ a rather low vibration rate, using vibrations of considerable force or amplitude, rather than very rapid vibrations to which the ear still responds with apparently normal ease. For the same reason, low musical tones may of themselves be of value in exercising the ossicles in cases of beginning tympanic deafness.

From the foregoing pages it will be seen that the writer has made no attempt to describe an inelastic course of treatment which would fit all, or even a majority, of these cases. He has tried rather to describe certain morbid conditions which may be met with in either the hypertrophic or hyperplastic form of chronic otitis media, and to suggest the more rational means for correcting the same.

I believe that we should not make use of too many therapeutic agents simultaneously, since by so doing we shall inevitably mask the results of the different agents employed. For example, in a foreign clinic the writer once saw a considerable number of patients under treatment by a galvanic current of moderate strength applied alternately in front of and behind the ear for a period of five or ten minutes. To determine the value of such treatment, other measures should for the time being be excluded. If at one sitting we inflate the tympanum, pass a bougie for purposes of tubal dilatation or massage, and supplement these measures by some form of external vibratory massage, we shall certainly be at a loss in analyzing our results,—whether of functional gain or loss. I believe, therefore, that if we are endeavoring to restore the normal calibre of a restricted tube, it is best to postpone other local treatment until we are satisfied with the mechanical widening of the tube, and are able to determine what functional gain, if any, has resulted from this change. Following this plan, and weighing results by frequent hearing tests, we shall soon know definitely (1) whether the patient is being benefited, and (2) to what particular agent a demonstrable improvement is due.

Before leaving this subject, a word must be said as to the constitutional or general management of these cases. Undoubtedly some cases of chronic middle-ear catarrh have a constitutional basis in subacute or chronic gout or rheumatism. When such a diathesis can be determined, the careful physician will recognize its possible bearing upon the tympanic lesion and the importance of constitutional remedies.

In a majority of cases it is difficult or impossible to trace any relation between the aural disease and any constitutional disorder. The general treatment often narrows itself, therefore, to a regulation of the patient's mode of life,—this including such minutiæ as clothing, diet, use of tobacco or alcholic drinks, habits of open-air exercise on the one hand or of over-

work and excessive confinement to office or business on the other. These influences vary so greatly in different cases that they can not be disposed of dogmatically. In the case of tobacco, for example, we know that many excessive smokers go through life without discoverable ear lesion or disorder. When, therefore, a patient with chronic aural disease acknowledges to a moderate use of tobacco, I believe that we should investigate rather carefully its possible influences before depriving him of a comfort for which a more harmful substitute may be found. If we believe that even moderate smoking is in a given case harmful to the individual, it should undoubtedly be advised against. Excessive smoking is always harmful to these patients, either in its effect upon the tubal mucosa or in the influence of the tobacco upon the auditory nerve.

Since all these matters must be discussed more at length in connection with otosclerosis, the student is urged to a careful consideration of the treatment of that disease.

OTOSCLEROSIS.

While no pathological relationship between otosclerosis and disease of the middle ear has been demonstrated, the clinical resemblance is in many cases so great as to render a differential diagnosis no easy matter. It seems best, therefore, that it be considered side by side with the other lesions commonly leading to impaired hearing or deafness.

Definition.—The term otosclerosis is employed to describe the condition in which, independently of the health or intercurrent disease of the tympanum, the bony capsule surrounding the labyrinth is the seat of chronic non-suppurative disease interfering with the function of hearing.

Etiology.—With reference to the etiology, it is well to acknowledge at once that we have as yet no definite knowledge as to the exciting causes of this disease. It has occurred with sufficient frequency in syphilitic subjects to lend some weight to the conjecture that syphilis in some way influences its inception. Habermann[6] is among those who have held that syphilis is a direct cause of the disease. Körner,[7] on the other hand, in analyzing the grounds for this belief, could find therein no proof that otosclerosis is ever of syphilitic origin. Professor Denker,[8] of Halle, made a thorough and careful investigation of 27 cases in which the clinical histories were recorded during life and in which the tissues were examined post mortem under the microscope. As a result of these studies and a review of the literature, Denker concludes that there is no proof that syphilis is ever a direct cause of otosclerosis. It now seems probable that syphilis is a factor in the progress of the lesion rather than in its causation. Thus, otosclerosis in an individual suffering from constitutional syphilis is said frequently to run a rapid course leading early to marked deafness. Naturally gout and rheumatism are present in a certain proportion of

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[6] Habermann: Arch. f. Ohrenh., Bd. lx.

[7] Körner: The Heredity of Otosclerosis, Arch. of Otol., Amer. Edition.

[8] Denker: Die Otosklerose, p. 81–84.

cases, but if they have any causal relation to the disease, it is probably an indirect one exerted through the anæmia and lowered physical state to which they give rise.

While the disease is sometimes seen in patients who also present evidences of tympanic disease, it seems probable that the two lesions are in most cases quite unrelated. Certainly many cases of otosclerosis have been observed in which there was complete absence of all signs of tympanic disease, past or present. As to whether tympanic disease is ever the direct cause of the lesion under discussion, there is still some difference of opinion, with the consensus of opinion steadily turning to the belief that otosclerosis is a pathological entity never secondary to a tympanic lesion. This question will be referred to later.

An etiological factor which heretofore has received scant attention, but which is destined to have greater consideration in future, is the influence of poisons conveyed through the blood stream from extraneous foci of infection. If infected tonsils, carious teeth, or pus foci in any part of the body may cause inflammatory changes in the articular structures of the larger joints, why not also in the joint structures of the ossicular chain? And why may not the same metastatic poisons influence—at least in some cases—the initial changes in the bony labyrinthine capsule leading eventually to the characteristic features of otosclerosis? I believe that the tonsils are destined in future to come more frequently, and I hope earlier, under suspicion in their possible relation to otosclerosis.

While it is conceded to be a disease of middle life, usually commencing after the twentieth year, Körner has shown that it may occur much earlier, recording two cases from his own practice in which the disease was present at ten years and fifteen years respectively. Sex seems to influence predisposition to the extent that women are more frequently sufferers than men,) *e.g.*, 58.2 per cent. in women, 41.8 per cent. in men (Denker). Heredity undoubtedly plays an important part in the spread of the disease, as has been proved by Hammerschlag, Körner, Gray, and others, who have published family trees showing remarkable reproduction of the disease.

Morbid Changes Affecting the Mobility of the Stapes.—In the minds of many, the association of otosclerosis with bony fixation of the stapes is a fixed idea; hence the determination in any case of stapedial ankylosis is apt to suggest the presence of otosclerosis. It is obvious that such deductions may lead to faulty diagnosis.

Firm fixation of the stapes in the oval window or niche may be caused by many conditions,—as, for example, the fibrous bands sometimes resulting from chronic hypertrophic otitis media, which may give rise to a very high degree of stapedio-vestibular ankylosis. Such processes have been very clearly demonstrated by Politzer [9] and others. Another form of stapedial fixation occasionally results from middle-ear suppuration, in which masses of granulation tissue occupying the oval window recess may be transformed first into fibrous tissue, and later as a result of inflammatory

[9] Politzer: Diseases of the Ear, p. 278.

changes in the surrounding bone may even be converted into bone (Pritchard).[10] It is needless to say that such conditions have no relation to true otosclerosis.

Pathology.—Otosclerosis is a lesion originating, so far as we know, in the bony capsule surrounding the labyrinth, and varying in its mechanical results according to the region chiefly involved. Occurring in the region about the oval window, it may give rise to ossification of the annular ligament, or rather to its absorption and replacement by bone, with resulting osseous fixation of the stapes. In other recorded cases, the morbid process has involved intra-labyrinthine structures,—*e.g.*, the spiral membrane, —and thus caused deafness without producing stapedial fixation. Furthermore, since foci of the disease have been observed in widely separated portions of the petrous bone, there can be no doubt that characteristic changes may occur in parts of the labyrinthine capsule not closely related to structures essential to the cochlear function, in which case the patient would still have otosclerosis, though a diagnosis would not be made on account of the fortunate absence of symptoms.

The morbid process seems to lack any of the characteristic features of an inflammatory process. It consists essentially of the absorption of the old bone in the region involved and its replacement by new spongy bone. The new spongy bone is characterized by Havarsian canals of abnormal width, and by large medullary spaces containing numerous multinuclear cells and large thin-walled blood-vessels. The new bone is also characterized by its strong affinity for hæmatoxylin and carmine dyes. It is differentiated by a distinct line of demarcation from the surrounding healthy bone. It may retain its spongy character, or may in time be converted into dense bone. These changes may occur in any part of the bony capsule of the labyrinth. The commonest point of attack is that portion of the labyrinthine capsule just above and in front of the oval window. Occurring in this region, the upper and anterior margins of the window undergo the characteristic changes. Absorption of any portion of the margin of the oval window cuts off the nutrient supply of the contiguous portion of the annular ligament of the stapes (Gray). The ligament, therefore, is also absorbed, its place being occupied by new spongy bone, which later may be converted into compact bone.

The foregoing paragraph appeared in the first edition of this book and is as pertinent today as when it was written. In his more recent work on otosclerosis, Gray discusses the origin and pathology of the disease more from the biologic viewpoint. Drawing upon the storehouse of his extensive knowledge of comparative anatomy, he pictures for us the gradual evolutionary development of the labyrinth. From its most elementary type in certain primative fish forms, the labyrinth, then embracing only a rudementary vestibular apparatus, is gradually evolved, finally reaching in the more highly organized fishes the most highly developed and complex form of vestibular mechanism. Only with the appearance of the amphibia

[10] Pritchard: Fixation of Stapes, Trans. Otol. Society United Kingdom, Vol. vii, p. 60.

FIG. 143.—Horizontal section through oval window (right), dividing stapedial foot-plate, vestibule, and basal turn of cochlea. (After Paul Manasse.)

A, newly formed bone in region of anterior margin of oval window; *B*, foot-plate of stapes.

Stapes itself is not involved, nor is the annular ligament destroyed. Stapes is therefore not ankylosed. Coincidently there is complete atrophy of Corti's organ.

Fig. 144.—Horizontal section through stapes and basal turn of cochlea. (After Paul Manasse.) The characteristic bone changes are seen in the foot-plate of the stapes, extending thence in front of anterior border of oval window through the entire thickness of the cochlear capsule to its inner periosteal lining.

The posterior end of the stapedial foot-plate is enormously thickened by the outgrowth of the new bone and the annular ligament is destroyed, giving rise at this point to complete osseous ankylosis. At the anterior margin of the oval window, the annular ligament, though bounded on either side by newly formed bone, still persists.

The auditory nerve and spiral ganglion are atrophied.

does the adjunct mechanism of hearing, the cochlea, begin to appear. From this we see that the vestibular and semicircular canal system is older by untold ages than the cochlear mechanism. This fact, as will be shown later, may be not without bearing upon the pathogenesis of otosclerosis.

Gray [11] regards otosclerosis as a degenerative process, in which evidences of inflammatory changes, past or present, are completely absent. It is, moreover, according to Gray's view, a degenerative process dependent upon certain inherent defects in the bone cells or bone structure of the organ of hearing. Regarding such structural defects or errors as developmental variations, and recalling that such variations are particularly prone to involve structures of comparatively recent biological descent, we are provided with a hypothesis by which to explain the frequency of sclerotic changes in the cochlea, and their comparative rarity in the structures of the vestibular mechanism.

Gray apparently believes that these biologic or developmental defects in the labyrinthine capsule play a major part in the prevalence of the disease. If the developmental defects are sufficiently pronounced, the individual is not likely to escape otosclerosis; if they are completely absent, no exciting cause will induce the disease; between these two extremes are individuals who may or may not escape the disease, and in whom intercurrent conditions—*e.g.*, acute otitis media, toxic agents in the blood, syphylis, etc.—may be the deciding factor.

With Dr. Gray's view as to the incidence of the disease, Dr. J. S. Fraser seems not to be in complete accord in that he apparently believes that an exciting cause of extra-labyrinthine origin is necessary to its inception; and among such causes he gives acute inflammatory lesions of the middle ear a very important place. This view, whether accepted in whole or part, calls for serious consideration, on account of its important bearing upon the question of prevention or prophylaxis.

That the process is one of absorption of the normal bone and its replacement by new spongy bone, rather than rarefaction or spongification of the old bone, is clearly shown by the accompanying illustrations (Figs. 143, 144, 145). Two of them represent cases of otosclerotic stapedial ankylosis. In each it is seen that the new spongy bone occupies not only the position of a previously existing normal structure, but extends far beyond its normal limits. It is clear, therefore, that the lesion may give rise to exostoses from different parts of the bony capsule. These growths may protrude externally from some aspect of the promontory encroaching upon the tympanic space, or internally into the vestibulum. Extending from one or both walls of the oval niche, this recess may be so narrowed as to lock the stapes in an immovable grip. Or the cartilage covering the foot-plate of the stapes may be absorbed, and its place occupied by a bony growth extending into the vestibular cavity (Fig. 144). Obviously stapedial fixation may be produced in two ways,—*i.e.*, (1) by osseous union to the

[11] Gray: Otosclerosis; H. K. Lewis & Co., London, 1917. Gray: Otosclerosis; Review of J. S. Fraser: Journal of Laryng. and Otol., Vol. XXXIII, p. 16.

margins of the oval window, and (2) by pressure of bony growths from other parts of the labyrinthine capsule (Fig. 143). Politzer has recorded a case in which, in addition to characteristic changes involving the stapes and oval window, the scala vestibuli was completely filled by compact bone. Contemplation of the character of the lesion is enough to convince one of the futility of local treatment.

The causes and conditions which inaugurate these changes are still a fruitful subject of investigation. Siebenmann's investigations led him to believe, as Gray now holds, that otosclerosis is a developmental abnormality rather than a disease in the ordinary sense. He regards it as a last stage in the process of the development of the labyrinthine capsule,—a stage which ordinarily does not take place. He believes that the process originates not in the bony capsule, but in the layer of cartilage-cells lying between the original membranous capsule of fetal life and the surrounding bony capsule later developed from the connective tissue of the skull. According to this view, otosclerosis is in reality a process which, while abnormal in the labyrinthine capsule, is the rule in the other bones. To understand this theory we must remember that the bony labyrinth attains approximately its maximum size at birth. This, of course, is the reverse condition to that governing the other bones of the skeleton, which from birth to maturity must constantly increase in size while maintaining their presribed form.This process of growth is accompanied by a continual loss and regeneration of tissue. Similar changes occurring in the fixed and closely confined bony labyrinth cannot fail to result in distortion or obliteration of normal structures. Siebenmann's theory, while never generally accepted, seems to have commanded the respect of Körner and many other distinguished students of otology.

Apparently we are on the threshold of a period of renewed interest and activity—at least throughout the Anglo-Saxon world of otologists—in the study of the causes and pathogenesis of this at present rather hopeless disease. Unfortunately the enormous amount of work which in the past has been focused upon the pathology of otosclerosis, has yielded results of academic interest, rather than of practical value to suffering humanity. We know (something of) the histologic character of the lesion, and we are provided with fairly convincing theories as to the pathogenesis; but as to treatment or prevention we have only hypothesis based for the most part on pure conjecture. There is nothing in the way of evidence that any causal relation exists between tympanic lesions, inflammatory or suppurative, and the characteristic bone changes of otosclerosis. While such hypothesis may furnish useful bases for a perfectly justifiable empiricism, they may never-the-less be misleading and may involve the disadvantage of retarding work in a direction of effort to establish a more solid basis for treatment, preventive or corrective. In the solid work of Gray, Siebenmann, Manasse, Körner and others we have the ground work for the next advance.

A more recent hypothesis is that advanced by Gray, of Glasgow,[11] who connects the lesion with failure of the local blood supply, and in this way brings it into some etiological relation with depressed systemic conditions, and particularly with those in which anæmia is a prominent feature.

Fig. 145.—Horizontal section through right oval window. (After Paul Manasse.)

Widespread and characteristic bone changes are seen involving the foot-plate of the stapes, the entire thickness of the cochlear capsule, and the outer wall of the vestibule. The more lightly stained areas represent the changes of long standing, the dark areas the more recent proliferation of new bone.

Total destruction of annular ligament and complete osseous ankylosis of stapedial foot-plate are seen at posterior margin of oval window; the same process, though in a less advanced stage, being in evidence at the anterior margin of the oval window.

The membranous labyrinth and both branches of the auditory nerve are atrophied.

In support of this view, Gray marshals the following facts: (1) the well recognized absence of any evidences of inflammatory action; (2) the character of the initial change,—*i.e.*, absorption of bone and cartilage throughout small circumscribed areas of bone, which are differentiated by

[11] Gray: Transact. Otol. Soc. United Kingdom, vol. vii, pp. 76–79

well-marked lines of demarcation from surrounding healthy bone; (3) the deposition of new spongy bone to replace the structures which have been absorbed; (4) the fact that this absorption without inflammatory reaction and the subsequent replacement by spongy bone are what might be expected in a tissue in which the local blood supply had failed; (5) the character of arteries supplying bone,—*i.e.*, that of vessels incapable of changes in calibre in response to variation in arterial force; and, finally, (6) the anatomical peculiarities of the bony capsule,—*i.e.*, very dense bone in which the calibre of the Haversian canals is much smaller than in any of the other bones of the body. This reduced calibre of the Haversian canals increases the friction which the heart action has to overcome. Gray, therefore, believes that, when for any reason the arterial force is markedly reduced, and particularly if the quality of the blood is pathologically changed, the conditions become ripe for stagnation of the blood in the vessels traversing the bony capsule, with possibly resulting thrombosis. Thrombosis in these minute vessels effectually cuts off the blood supply from certain circumscribed areas of bone, in which consequently are inaugurated the characteristic changes of the disease. Gray holds that this theory explains in part the greater predisposition of women to the disease, women being more subject than men to various forms of anæmia. It does not, on the other hand, coincide with the strong influence which heredity is known to exert upon the incidence of the disease.

Symptoms and Diagnosis.—All observers agree that a perfectly uncomplicated case of otoslcerosis, with bone changes confined to the stapedio-vestibular region, is easily diagnosticated. There are in the main but two varieties of the disease which present special difficulties of diagnosis,—namely: (1) otosclerosis with foci of disease so distributed as to involve directly the cochlear structures, and (2) otosclerosis complicated by chronic catarrhal otitis media. We shall begin, therefore, with the symptoms which characterize the lesion in the most uncomplicated form, —viz., otosclerosis with deafness dependent upon fixation of the stapes.

History.—In characteristic cases there is usually a history of very gradual development of symptoms. Frequently there are no intercurrent diseases or disorders with which the patient associates his aural complaint. If the lesion is advanced, both ears are almost invariably involved. According to Bezold, the lesion is bilateral in 88 per cent. of cases. In the early stages, however, evidences of the disease may be confined to one ear. Frequently the ear in which symptoms appear last is the one in which the progress of the lesion is subsequently most rapid and the deafness or impairment most pronounced. The history may be absolutely negative as to catarrhal or suppurative conditions having involved the tympanic cavities. In a word, there may be no account of any conditions within the nose or nasopharynx, or in the Eustachian tubes, with which the aural lesion can be in any way associated.

Symptoms.—The patient usually complains of two symptoms,—namely, impaired hearing and tinnitus aurium. Either one may be the first to

attract his attention, though impairment of hearing is probably always the first symptom. It is conceivable that an adventitious subjective sound might disturb the patient while moderate recession of the normal hearing power might escape his notice. This probably explains the occasional statement that tinnitus was present long before the hearing became impaired.

Deafness.—The impairment of hearing is frequently so gradual in its advance as to be fairly pronounced before the patient is fully aware of his functional loss. In the early stages the impairment of hearing may be manifested by slight loss of auditory acuteness for the watch, acoumeter, and the conversational voice, and by very slight increase in hearing by bone conduction. Thus, the sound of a vibrating tuning-fork in contact with the mid-line of the skull may seem louder in the ear first affected, or in the one in which the lesion is more firmly implanted.

In advanced stages of the disease, the hearing tests give the following fairly definite reaction: The hearing distances for the watch, the acoumeter, and for the whisper and conversational voice are very considerably reduced. The lower tone limit is always elevated, and loss of hearing for progressively higher tones in the musical scale usually advances with the progress of the lesion. The perception of the higher musical tones is, on the other hand, well maintained, and the upper tone limit may be absolutely normal. The period of hearing by bone conduction is always definitely, and usually very considerably, prolonged.

This brings us to Bezold's triad symptom complex,—viz., (a) loss of hearing for the lower musical tones, (b) prolonged period of hearing by bone conduction, and (c) negative Rinné. Bezold claimed that the presence of this syndrome, in cases presenting no evidences either of tympanic disease or of disease of the auditory nerve, could be positively relied upon as pointing to otosclerosis with stapedial fixation.

As a corroborative measure, Gellé's test should be applied. If condensation of the air in the auditory meatus exerts no influence upon the intensity with which sound is heard through the cranial bones, the diagnosis of stapedial fixation is strongly confirmed.

Tinnitus Aurium.—There is no aural disease in which the subjective sounds are more constant or persistent. Fortunately, there are many cases in which the patient is apparently not greatly disturbed by them,—either by reason of their moderate intensity or from the fact that custom and possibly a strong will enable him to ignore them. In other cases the head noises are the most distressing feature of the disease, sometimes rendering life a burden. Unlike the impairment of hearing, the head noises are, fortunately, subject to periods of comparative amelioration, or at least to periods during which the patient is less disturbed by them. There is, however, no promise that they will disappear as the disease advances; and if the lesion is confined to the region of the oval window, they are not unlikely to increase as the hearing power recedes. This, in the writer's opinion, constitutes one of the most alarming possibilities of

this dreadful disease,—a possibility which with nervous or mentally unstable individuals may far outweigh the serious one of prospective loss of hearing.

Vertigo.—While vertigo is not a particularly prominent feature of the disease, moderate dizziness is complained of in a certain proportion of cases (22 per cent., Bezold). It usually does not amount to more than slight subjective vertigo, lasting but a few moments. In others, it assumes so severe a character as to overbalance all other symptoms, and to justify even the most drastic surgical measures for its relief (Lake).

Paracusis Willisii.—This condition has already been described as an occasional symptom of hyperplastic otitis media with fibrous fixation of the stapes. It is a much more frequent phenomenon of otosclerosis unaccompanied by disease of the auditory nerve.

The next step is the examination of the ears both by inspection and by catheter inflation. Inflation usually reveals a tube of normal calibre, and is practically without influence upon the deafness,—*i.e.*, no noteworthy improvement results as in the case of chronic catarrhal otitis media. This is an important diagnostic point. If, in addition to this, the drum membranes are seen to occupy approximately their normal position and to present no changes characteristic of chronic middle-ear disease, the diagnosis of otosclerosis may be regarded as proved.

There is one physical sign which is regarded as more or less strongly indicative of otosclerosis,—viz., the presence of a reddish or pink blush, seen upon, or rather through, the membrana tensa, usually behind the umbo (Schwartze). It is in many cases absent. When present it is supposed to be due to congestion of the mucous membrane covering the promontory, secondary to pathological changes in the bone beneath.

Before leaving this subject the writer wishes to revert for a moment to a consideration of Bezold's syndrome,—viz., prolonged hearing by bone conduction, elevation of the lower tone limit, and reversed ratio between hearing by air conduction and bone conduction. This combination is present also in any tympanic lesion giving rise to stapedial ankylosis. It is, therefore, of special value as pointing to otosclerosis only in cases in which tympanic disease can be definitely excluded. Since disease of the auditory nerve regularly results in reduction of hearing by bone conduction, Bezold's syndrome must be lost or changed whenever the lesion involves the cochlear filaments of the nerve. It is, therefore, of considerable value in determining whether the lesion is confined to the stapedio-vestibular region or is so distributed as to involve also the cochlea.

To epitomize: The characteristic features of the disease may be summed up as follows: History of hereditary predisposition (frequently obtainable); history of very gradual, but progressive, bilateral impairment of hearing, not subject to marked variations in response to external conditions; tinnitus present and usually persistent; paracusis present; absence of physical signs of chronic tympanic disease; patent Eustachian tubes; catheter inflation followed by little or no real functional gain. Deafness

characterized by auditory failure for low musical tones, upper tone limit remaining normal or nearly so; bone conduction increased; Rinné negative. Intensity of sound as conveyed through the cranial bones uninfluenced by compression of air in auditory meatus (Gellé). With such a picture the physician who fails to recognize otosclerosis with stapedial fixation is certainly not a practical otologist.

Otosclerosis with Involvement of the Cochlea.—In describing the pathology of otosclerosis, it was pointed out that the disease may attack any portion of the labyrinthine capsule; and that while the region of predilection is found in the immediate neighborhood of the stapedio-vestibular articulation, the lesion may be so distributed as also to involve the intracochlear structures. In this way may be inaugurated changes affecting the cochlear nerve filaments, leading to many of the symptoms characteristic of a primary nerve lesion. Obviously such extension of the disease must produce some modification of the clinical picture.

Symptoms.—For emphasis and comparison, let us consider first a case of otosclerosis with extensive involvement of the cochlea and in which fixation of the stapes has not taken place. As in the type first described, the patient usually complains of impaired hearing in both ears, and subjective noises. The deafness is at first very gradual in its advance, but later may be subject to very marked periodical changes for the worse,—*i.e.*, loss of hearing which is never thereafter regained. The tinnitus is usually a distressing symptom. It may be very persistent, or may become less severe with the progress of the disease, finally subsiding as the patient approaches absolute deafness. This, however, is not invariable, cases having been recorded in which patients have become absolutely deaf yet continued to suffer from the most distressing head noises. Paracusis Willisii is not present. There may be no history of past or present tympanic lesions having any etiological relation to the disease.

Examination of the ears reveals comparatively normal drum membranes,—*i.e.*, drum membranes fairly normal in position and showing no marked structural changes. The characteristic red "blush" behind the umbo may or may not be present. The Eustachian tubes are patent, and catheter inflation exerts no influence on the hearing.

Deafness.—In advanced stages the hearing tests show the following characteristic changes: The lower musical tones may be well perceived and the lower tone limit but little changed or practically normal. The upper tone limit is lowered, and all the higher tones are heard with reduced intensity. The period of hearing by bone conduction is reduced, and Rinné remains positive or normal. Bezold's triad syndrome is therefore absent.

Hartmann's Differential Point.—A rather striking differential test, based upon the normal hearing periods for the different tuning-forks by aerial conduction, was first announced by Ed. Hartmann. He found in lesions involving the cochlear nerve not only that the higher tuning-forks were heard during a much shortened period, but also that the relative

curtailment as compared with the normal hearing periods become progressively more marked as we ascend in the musical scale. Exactly the reverse is the case with otosclerosis confined to the stapedio-vestibular region. The experiment may be stated briefly as follows: In otosclerosis confined to the stapedio-vestibular structures, the hearing periods are greatly reduced for the low musical tones, but gradually approach the normal as we ascend in the musical scale. In otosclerosis with involvement of the cochlear nerve (the stapes remaining mobile), the hearing periods are progressively reduced as we ascend from the lower to the upper end of the musical scale.

In the above we have taken two extreme types of the disease in order to bring out more clearly and graphically the clinical features characteristic of stapedial ankylosis on the one hand, and disturbance of the cochlear nerve on the other. In a majority of cases, however, in which post-mortem findings have demonstrated the presence of intracochlear disease, there have been coincident changes in the region of the oval window resulting in fixation of the stapes. It is clear, therefore, that we shall find in many cases functional reactions characteristic both of stapes ankylosis and of disease of the cochlear nerve. Thus, both upper and lower tone limits may be curtailed, and the tendency to increase in bone conduction commonly resulting from stapedial fixation may be counteracted by the cochlear lesion. With such contradictory reactions, it may be difficult to differentiate between otosclerosis and a lesion primarily involving the cochlear branch of the auditory nerve. As bearing upon such a problem, a possible history of heredity, a history of gradual bilateral onset, and the determination by functional tests of greater disturbance of hearing for the lower musical tones than would ordinarily result from a primary nerve lesion, may aid us in reaching a correct diagnosis.

The course of the disease varies greatly in different cases. In some cases the disease progresses rapidly from the start, leading within a comparatively short period to very marked—sometimes to profound—deafness. Such rapid advance in the lesion and its resulting deafness has been observed to occur with greater frequency in patients suffering also from constitutional syphilis. Politzer states that women suffering from otosclerosis often show a considerable permanent increase in the deafness with each childbirth. When, without intercurrent constitutional disease, the aural disease runs a particularly rapid course, the inference seems admissible that the lesion is so distributed as to involve early the perceptive mechanism proper. Fortunately, there is a large class of cases in which the disease is very gradual in its advance. In some cases years may elapse without apparent progress in the lesion. To this extent, however, the prognosis is always bad,—namely, that individuals in whom the lesion has produced marked impairment by middle life will probably in old age reach a very distressing grade of deafness. The most hopeful prognosis belongs to those cases in which the disease develops late in life, or rather in which the functional disturbance then demonstrable is of moderate grade. Such a case

may be so gradual in its advance as to bring the patient to old age without disabling deafness.

Otosclerosis Complicated by Chronic Catarrhal Otitis Media.—When chronic catarrhal otitis media coexists with otosclerosis, the one lesion may so obscure the other that a positive diagnosis of the two conditions can not be made. Certainly such a diagnosis is quite impossible as a result of a single examination, no matter how careful this examination may be. As pointing to otosclerosis we may in some cases have the following somewhat indefinite facts,—viz., (1) the deafness may be more pronounced than would be expected from the tympanic lesion alone, and (2) local treatment may exert less influence upon the symptoms than would be expected in uncomplicated tympanic disease. With well-marked evidences of tympanic disease the early stages of otosclerosis can not be diagnosticated.

The opinion expressed by Whiting, that profound deafness is always due to labyrinthine or nerve disease, is a belief probably subconsciously held by most otologists. With respect to tympanic disease, there is a degree of deafness which probably is never reached unless the movements of the stapes within the oval niche or window are mechanically restrained. Personally, I believe that a reversed Rinné always means stapes fixation. But with the physical evidences of chronic non-suppurative tympanic disease, whether a negative Rinné points to fibrous immobilization or to osseous union resulting from otosclerosis, is a question which is not always soluble.

Treatment.—If we accept Siebenmann's theory that the disease is simply an abnormal stage of development, all treatment seems useless. If we agree with Gray that the disease may be in some degree dependent upon depressed systemic conditions giving rise to anæmia, the prospect seems more favorable. In its most typical form,—*i.e.*, otosclerosis without evidences of tympanic disease,—I can see no possible influence which local therapeutic measures can exert upon the progress of the lesion. The occasional statements of well-known otologists as to cases of otosclerosis which have responded favorably to local treatment are probably to be regarded as cases of mistaken diagnosis. Such experiences are not, however, to be altogether ignored, since they serve to emphasize two facts,—viz., (1) that cases of deafness apparently traceable to otosclerosis are sometimes on further investigation found to be cases of temporary deafness dependent upon transient causes, which yield readily to local treatment; and (2) that the deafness of otosclerosis is sometimes markedly increased by certain obscure but transient conditions within the tympanum, such cases showing rapid improvement as a result of local treatment. As illustrating the class just referred to may be mentioned the following experience of the writer: A patient referred to him by another physician gave a history of slight impairment of hearing of two or three years' standing which had recently become very marked in the left ear. Both drum membranes were practically normal in appearance. Functional tests showed

in the left ear a type of partial deafness characteristic of disturbed sound conduction. Little or no improvement resulted from the first inflation. As a result of these findings a tentative diagnosis of otosclerosis was made, the patient being advised to submit to a short course of treatment in order to prove definitely the character of the disease. To the writer's surprise, the hearing very rapidly improved under the plan of treatment usually employed in chronic catarrhal otitis media, soon reaching a degree of acuteness which, though slightly below the normal standard, might be regarded as little more than the physiological impairment due to his fifty-five years. In this case the writer did not flatter himself that he had relieved a case of otosclerosis, but rather that he had relieved some transient condition within the tympanum, the nature of which he had not been able to determine.

Leaving such exceptional cases out of our consideration, we must come back to the regrettable fact that otosclerosis is a lesion which ordinarily is not helped by local treatment of any kind. Catheter inflation usually reveals open tubes and to some extent exercises the drum membranes, but can have no influence upon the progress of the disease. Politzer states that vibratory massage through the external auditory meatus is of value in the early stages of the disease,—*i.e.*, before osseous union between the foot-plate of the stapes and margin of the oval window has taken place. He also recommends the administration of iodide of potassium in 5-grain doses three times a day, this to be continued through 25 or 30 days, and to be repeated two or three times during the year. Politzer believes that this periodic use of the iodides helps in beginning otosclerosis to stay the progress of the disease.

Siebenmann advocated small doses of phosphorus, to be given more or less continuously over a period of years. It is worthy of note that neither the iodide of potassium nor the phosphorus was expected to result in improvement of hearing, but simply to retard the progress of the disease. Couple this fact with the statement of Bezold that a large number of cases become stationary with only moderate impairment of hearing, and we have a commentary on the difficulty of determining the real value of these or other drugs in this disease.

While otosclerosis is a disease for which local treatment promises little, there is much that can be done for such a patient. In the first place, we are able in many cases to assure him that his deafness will in all probability be very gradually progressive, and that it may never reach a degree interfering seriously with his usefulness. There is no question that much needless suffering has been caused in certain cases by a frankly unfavorable prognosis, the correctness of which has been disproved by subsequent events. The writer has under his care a patient for whom another aurist eight years ago predicted rapidly progressive loss of hearing. And yet during the past eight years this man's hearing has not changed demonstrably for the worse, and during this period he has been able to accomplish a very considerable amount of useful work. I believe, therefore, that when

the hearing is only moderately impaired, we should acquaint the patient with the nature of the lesion only to the extent of making him appreciate the importance of carrying out such general provisions as may seem best for him, and that their neglect carries distinct danger of serious deafness.

There is probably no disease in which hygienic measures and a wise regulation of the patient's mode of life are of more importance than in otosclerosis. This view seems admissible when we consider the following facts: It is known that constitutional syphilis, if not a cause of the disease, is at least a factor influencing its rapid advance; that anæmia influences its progress; that with women the onset of the disease seems frequently to be associated with the age at which they are most subject to chlorosis; that women suffering from otosclerosis commonly show a definite advance of the lesion, as evidenced by permanent increase in deafness, after each childbirth. Presumably these conditions influence the disease either through lowered vitality or through the anæmia which is their common characteristic. If these depressed constitutional conditions hasten its advance, it seems a logical deduction that by the reverse process, of building the patient up to his maximum of physical health, we may to some extent hold the disease in abeyance.

In the section dealing with the etiology, reference is made to tonsillar infection as a possible and probable factor in the incidence of certain cases of the disease. Naturally if such a causal relationship could be established in any large series of cases, the importance of tonsillectomy in the control of the disease would be evident. There is at hand no data justifying any statement more definite than that the tonsils should be carefully studied in the early stages of all cases of otosclerosis. Three cases reported by Mr. Richard Lake of London are of sufficient interest to justify their inclusion in brief outline here.[12]

Case I. Was that of a young woman, *æt.* 24, who presented characteristic symptoms of otosclerosis. Removal of the tonsils brought marked improvement, both in the hearing and as to the physical appearance of the drum-membranes. A relapse was traced to an infected maxillary sinus, disinfection and treatment of which resulted once more in marked auditory gain.

Case II. "Women, aged about 25, with recent history of rapidly increasing deafness; both ears showed red reflex. Tonsils removed without delay. Two weeks later, redness of fundi gone and hearing as good as ever."

Case III. Woman of thirty years, suffered from deafness diagnosticated as due to otosclerosis. Treatment by various methods resulted only in moderate and temporary improvement in hearing. Finally the tonsils were removed, with a gain in hearing which up to the time of the report had been maintained.

There is no doubt that overwork, mental or physical, the strain of business anxieties, excessive confinement to business, unrelieved by regular hours of exercise in the open air, may unfavorably influence the disease. As

[12] Richard Lake: Journal of Otology and Laryngology, vol. XXXVII, 1922, p. 361.

nearly as the individual's circumstances admit, it should be insisted upon, therefore, that his business activities be brought within reasonable limits, and he should be made to understand that any indulgence in such business excesses may in the end be paid for dearly by serious loss of hearing.

Tobacco and alcohol, when they influence the hearing injuriously, do so chiefly through their action upon the auditory nerve. It is clear, therefore, that excessive smoking or drinking may have disastrous results in a lesion in which the end organs of the nerve may be maintining their function under conditions which have already become abnormal. Tobacco and alcohol should, therefore, be indulged in only with the greatest moderation.

Drugs, other than such tonics as may be occasionally indicated, have little or no specific value in otosclerosis. The patient should be instructed that quinine, the salicylates, and most of the coal-tar derivatives induce intra-labyrinthine congestion, and are under ordinary conditions distinctly contra-indicated.

Gray places great emphasis upon the importance of combating anæmia in any form; and it may be that periodic blood examinations—particularly young women suffering from otosclerosis—might suggest measures which would prove of value in maintaining these patients in the best condition or combating the advance of the aural lesion.

Dr. J. A. Stucky of Lexington, Kentucky, believes an important factor in the progress, if not in the causation, of otosclerosis to be the circulation in the blood of toxins resulting from errors of diet. He has recorded his experience with a series of cases which he has had under observation during many years, and in which he is convinced that systemic care in preventing this form of auto-intoxication has played an important rôle in staying the progress of the disease.

The patient should be seen from time to time, and the hearing retested in order to determine the progress or the comparative quiescence of the lesion. It is of course particularly important that any catarrhal condition within the tubes or tympanic cavities should have prompt and efficient treatment.

The plan above outlined suggests in a general way what seems to the writer the best way of caring for these patients. While active local treatment is, as a rule, not indicated, it is important that the patient should consider himself constantly under the care of a competent aurist. By this is meant that he should return at stated intervals for examination and advice. This shifts the responsibility of his condition from his own shoulders, and, what is of considerable importance in these cases, eliminates the risk of much useless and possibly harmful treatment by ignorant or unscrupulous practitioners.

In what has been said above, the writer has had in mind the rather large class of cases in which the hearing is only moderately impaired, the lesion being apparently quiescent or very slowly progressive.

There is, unfortunately, another class of patients,—*i.e.*, those whose deafness is either progressing rapidly from bad to worse, or whose hearing is already so far lost as to render conversation exceedingly difficult or impossible. Such patients should be told frankly of the incurable nature

of their disease, and encouraged to make use of artificial aids to hearing. The very best advice that can be given to such a patient is that he apply himself diligently under the advice of a competent teacher to the task of acquiring the art of lip-reading. Only in this way can he rob his infirmity of its most unbearable hardship,—that of practical isolation among his fellows.

Surgical Measures for the Relief of Deafness.—Before leaving this subject, a word must be said as to the various surgical measures which have been proposed from time to time with the purpose of relieving ossicular ankylosis and thereby improving the hearing. They may be mentioned in the following order:

1. Mobilization of the stapes by passive movements by means of instruments introduced through a window in the drum membrane. The best opening is secured by a horseshoe-shaped incision in the posterosuperior quadrant (Fig. 146). The flap of drum membrane thus formed is then folded downward, leaving the incudostapedial articulation in view and accessible to instruments. The head of the stapes is then gently moved in different directions as far as its attachment to the lenticular process of the incus will allow without danger of injury to the annular ligament or of disarticulation from the incus. The results of this procedure have not given it a permanent place in aural therapy.

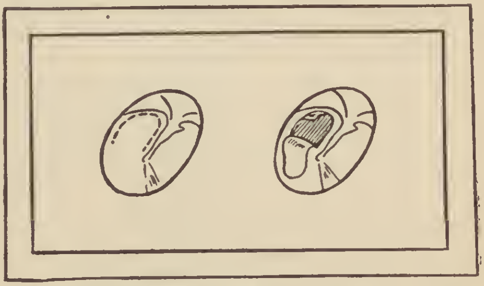

Fig. 146.—Diagrams showing line of incision, and the resulting flap, deflected.

2. Reduction of abnormal ossicular tension by division of the tendons of the tensor tympani and stapedius muscles. The fact that this operation has no champion among recognized authorities points to the disappointing results obtained.

3. Division of fibrous bands binding the crura of the stapes to the walls of the oval niche. Politzer makes the point that this operation, while without proved value in the fibrous fixation of chronic catarrhal otitis media, is in certain cases quite successful in the fibrous ankylosis following suppurative otitis media.

4. Removal of membrana tensa, malleus, and incus,—the stapes being left in situ. This procedure was based upon the hypothesis that the drum membrane, malleus, and incus, when incapacitated by disease for the function of sound conduction, should be removed in order that the stapes, acting alone, may be permitted to receive directly the sound waves from without and transmit them to the inner ear. The ultimate results of this operation proved so disastrous to the auditory function, that it has no advocates among the otologists of to-day. These operations, mentioned for their place in otological history, are quite obsolete today.

CHAPTER X.

THE ANATOMY AND PHYSIOLOGY OF THE LABYRINTH.

The inner ear, or labyrinth, embraces two distinct mechanisms,—viz., the *cochlea*, or essential organ of hearing, and the *vestibular apparatus* (saccule, utricle, and three semicircular canals), a contributory organ of equilibrium or orientation. These structures are contained within a series of little communicating cavities in the petrous portion of the temporal bone, known as the *osseous labyrinth*. Within the cavities of the bony labyrinth and surrounded by a supporting fluid, the perilymph, are the essential structures known as the *membranous labyrinth*. The inner cavities of the membranous labyrinth also contain a fluid which, to distinguish it from the surrounding perilymph, is called the endolymph. In form the membranous labyrinth follows rather closely the contour of the bony spaces in which it is contained.

Anatomically the labyrinth is separable into three main portions,—viz., a central cavity, the *vestibule;* an anterior portion, the *cochlea;* and a posterior superior portion, the *semicircular canals*. The vestibule lies just internal to the tympanum, with which, but for the foot-plate of the stapes, it would communicate by means of the oval window. Anteriorly it communicates with the cochlea, and posteriorly with the semicircular canals (Figs. 147 and 148).

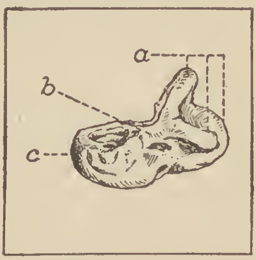

Fig. 147.

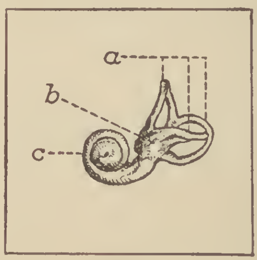

Fig. 148.

Fig. 147.—Bony capsule of labyrinth.
Fig. 148.—Drawn from metal cast of labyrinthine cavity. *a*, three semicircular canals; *b*, vestibule; *c*, cochlea.

The Osseous Labyrinth.—The cochlea consists of a bony tube measuring about 1½ inches in length, and coiled two and a half times about a central rod or cone into a form somewhat resembling a snail-shell. The diameter of the tube near its opening into the vestibule is about 2 mm., but from this point its calibre rapidly diminishes, so that after the first turn the average diameter is not more than 1 mm. The central axis of the cochlea—*i.e.*, from the centre of the base to the apex—lies in the horizontal plane and is directed forward and outward. Passing horizontally through the cochlea from base to apex is a central cone-shaped body known

as the modiolus. The modiolus is really a hollow central cone around which the spiral tube of the cochlea is coiled. Passing outward from the modiolus into the spiral canal of the cochlea is a lamina of bone called the *spiral lamina* (Fig. 149). The spiral lamina projects only half-way across the lumen of the cochlear canal. From its outer edge a delicate and important membranous structure, the basilar membrane, passes in the recent state to the outer wall of the cochlear canal, completing its division into two

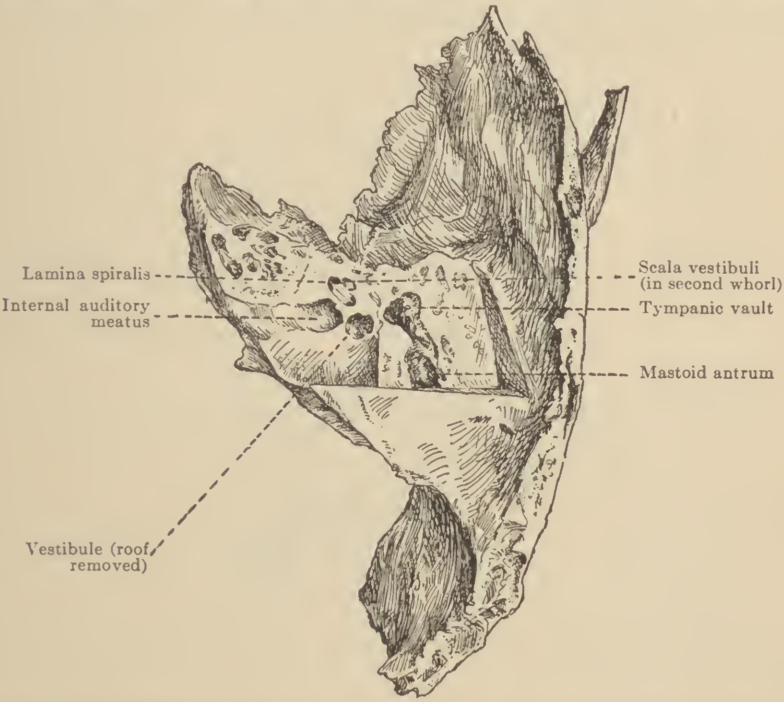

Fig. 149.—Horizontal section through internal auditory meatus, cochlea, and vestibule.

channels,—an inner, communicating with the tympanum through the round window, and known as the *scala tympani;* and an outer, opening into the vestibule, the *scala vestibuli*.

Note.—In most text-books the scala tympani is spoken of as the lower cochlear channel, and the scala vestibuli as the upper space. This method of description probably arose from the habit of illustrating the subject by diagrams of the cochlea with the apex uppermost. This, however, is not only incorrect but confusing to the student, whose effort is, and should be, to learn not only the relation of the various labyrinthine structures to each other, but also their relation to the various planes of the skull. With the head erect, the modiolus, representing the central axis of the cochlea, is not vertical but horizontal, and its direction from the vertical anteroposterior plane of the skull is horizontally forward and outward. The spiral cochlear tube winds around this horizontal body, and the spiral lamina, projecting vertically into its lumen, must necessarily divide it not into an upper and lower, but rather into an inner (basal) and outer (apical) space. This point is made clear by Fig. 150.

The scala tympani and scala vestibuli communicate with each other at the apex of the cochlear pyramid through a small opening known as the *helicotrema*. On the floor of the scala tympani, not far from its beginning at the round window, is a small opening leading into a short canal, the *aquæductus cochleæ*. This minute channel provides under certain conditions so dangerous a pathway of infection that the student should have a clear appreciation of its possible significance in disease. Beginning in the floor of the scala tympani near the round window, the osseous canal leads inward and slightly downward through the petrous bone to emerge by a

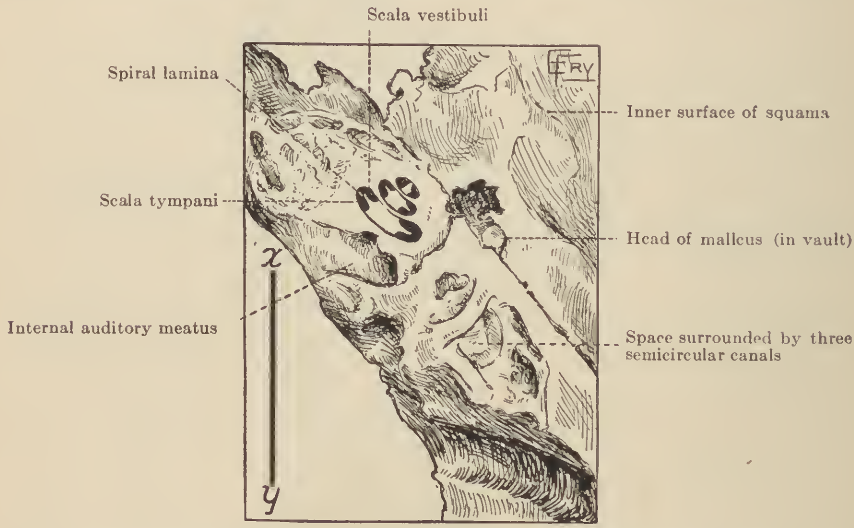

FIG. 150.—Horizontal sections through modiolus from base to apex (all structures enlarged). x—y, line giving direction of anteroposterior axis of skull.

triangular opening at the outer edge of the jugular foramen. The termination of the bony aqueduct is seen, therefore, on the basal surface rather than the interior of the skull. But while the bony aqueduct terminates at this point, the membranous canal is continued inward through the jugular foramen into the cranial cavity and perforates the dura to communicate directly with the subarachnoid space (Politzer, Schwalbe, A. A. Gray). According to Politzer, colored fluid introduced into the subarachnoid space quickly enters the cochlea and vestibule. The cochlear aqueduct is sometimes called *aqueduct of the perilymph*, because through it the cochlear perilymph may pass into the subarachnoid space. The important influence of this communication upon the possible consequences of a suppurative invasion of the labyrinth is obvious.

The central canal of the modiolus begins in a depression in the anterior wall of the internal auditory meatus near its fundus, called the fossa coch-

learis (Fig. 151), and gives passage to the cochlear branches of the auditory nerve. Surrounding the orifice of the central canal, the base of the modiolus (*i.e.*, fossa cochlearis) is perforated by numerous very minute foramina, leading into little canals which radiate outward between the bony layers, or plates, of the spiral lamina. These little canals receive the branches of the cochlear nerve, which in turn perforate the outer (apical) plate, or surface, of the osseous spiral lamina, to pass to their distribution in the basilar membrane and organ of Corti.

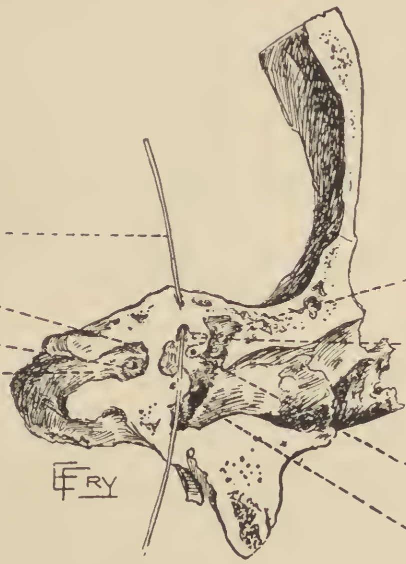

FIG. 151.—Vertical section through tympanum, vestibule, and internal auditory meatus.

The Vestibule.—The position of the vestibule in relation to the middle-ear cavity is indicated by the fact that the oval window opens into it and marks the centre of its outer wall. It measures 5 to 6 mm. in length (*i.e.*, from before backward), and approximately 4 mm. each in width and height. On the inner vestibular wall, and nearer the anterior than the posterior wall, is a slight ridge, approximately vertical in direction, known as the *crista vestibuli*. In front of this ridge is a small circular depression, the *recessus sphericus*, which in the living subject lodges the saccule. Behind the crista vestibuli is an elliptical depression, which lodges the upper part of the utricle, and is called the recessus ellipticus.

Openings into the Vestibule (Figs. 151 and 152).—The vestibule presents the five openings of the three semicircular canals. Each canal expands at one end into a bulbous enlargement known as the ampulla. The ampullæ of the horizontal and of the anterior vertical canals are situated near each other, these canals entering the vestibule through its superior wall, or roof, above the oval window (Fig. 151). The ampulla of the pos-

terior vertical canal is found in the floor (Fig. 152, *a*). Behind this—*i.e.*, on the posterior wall near the roof—is the opening of the small end of the horizontal semicircular canal (*c*). Further inward—*i.e.*, near the angle formed by the junction of the inner and posterior walls with the roof—is the common opening of the anterior and posterior vertical canals (*b*). In the anterior and outer corner of the vestibule,—*i.e.*, where the anterior

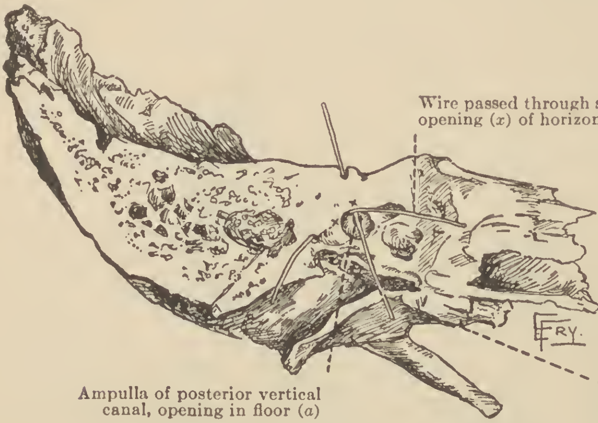

Fig. 152.—Labyrinthine vestibule (*d*) with roof removed.

and outer walls join the floor,—is the opening into the scala vestibuli. Still another minute opening is found on the inner wall of the vestibule leading into the aquæductus vestibuli. The crista vestibuli, recessus sphericus, and recessus ellipticus are perforated by numerous very minute foramina, constituting the so-called *maculæ cribrosæ*. They give passage to the saccular and utricular branches of the vestibular nerve.

The semicircular canals are about 1 mm. in diameter, except at their ampullæ where their calibre is about doubled. With the head erect and with chin indrawn, the horizontal, or external, semicircular canal lies very nearly in the horizontal plane. The two other canals are both vertical, the anterior vertical canal lying in a vertical plane directed from within outward and forward, and the posterior vertical occupying a vertical plane at right angles with that of the anterior vertical,—*i.e.*, directed outward and backward. It is obvious, therefore, that each canal lies in a plane at right angles to the other two (Fig. 153). In every case the ampullar end is situated further forward than the small end of the canal. In the case of the horizontal and anterior vertical canals this is made evident by a glance at any model or prepared specimen. With the posterior vertical, on the other hand, this fact is demonstrated only by tracing its lower, or ampullar, end to its opening in the floor of the vestibule, and there comparing its position with the common opening of the two vertical canals (Fig. 152). The posterior vertical canal is on a much lower plane than

the anterior vertical (Fig. 153). The anterior vertical canal is, therefore, by many authors called the superior vertical canal, the posterior vertical being called the inferior vertical canal. Anterior vertical and posterior vertical are, however, much more descriptive, and therefore more useful terms.

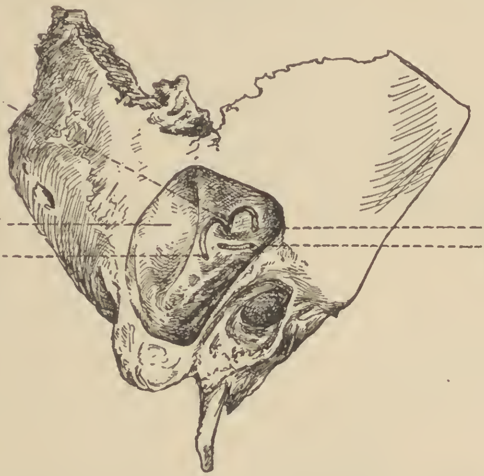

FIG. 153.—Relative positions of three semicircular canals.

THE MEMBRANOUS LABYRINTH.—The membranous labyrinth is everywhere partly surrounded by a supporting fluid, the perilymph. I say "partly" for the reason that it is in most regions connected at some point with the endosteum lining the walls of the osseous labyrinth. Owing to this intervening layer of perilymph, the various portions of the membranous labyrinth are necessarily much smaller than the bony spaces in which they are contained. The membranous semicircular canals, for example, are attached to the endosteum along the outer convex wall of the bony canals, being separated elsewhere by a considerable amount of perilymph. The membranous canals are, therefore, for the most part very much smaller than the osseous tubes in which they lie. Only at their ampullæ do they enlarge sufficiently to fill approximately their expanded bony compartments. Wherever in the labyrinth filaments of the auditory nerve perforate the bony capsule to reach the membranous labyrinth, the membranous parts so supplied are attached to the bone surface thus perforated, —*e.g.*, the cristæ acusticæ of the ampullæ and maculæ acusticæ of the utricle and saccule.

The membranous vestibule is found within the central cavity (vestibule) of the osseous labyrinth, and is partly surrounded by perilymph. It consists of the saccule and utricle and the vestibular structures by which they communicate with other parts of the membranous labyrinth. The saccule communicates directly with the scala media of the cochlea (ductus cochlearis) and only indirectly with the utricle. The utricle communicates by five openings with the three semicircular canals.

The utricle, about 5 mm. in length, is attached to the posterior part of the inner wall of the bony vestibule. Its upper half is lodged in the recessus ellipticus, behind the crista vestibuli. The portion of the utricle which rests against the recessus ellipticus is called the recessus utriculi. The recessus ellipticus is perforated by numerous small foramina for the passage of the utricular branches of the vestibular nerve; and where these nerve filaments enter the utricle, its inner wall is greatly thickened so as to project somewhat into the cavity of the utricular sac. This thickened area is covered by a highly specialized form of neuro-epithelium, and is known as the *macula acustica* of the utricle.

The cavity of the utricle communicates by five openings with the three semicircular canals (Fig. 154). The ampullar ends of the horizontal and

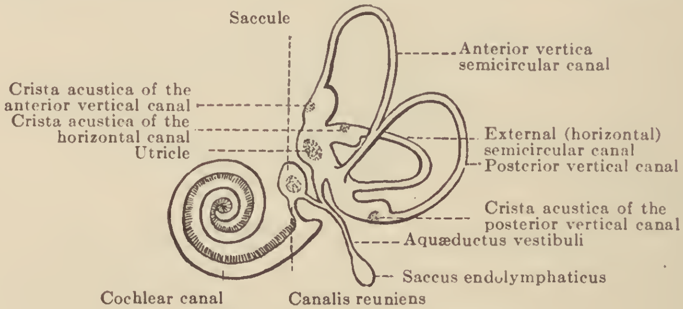

Fig. 154.—Membranous labyrinth (after Schäfer).

anterior vertical canals open into its roof; the ampullar end of the posterior vertical perforates the utricular floor, and the small opening of the horizontal canal and common opening of the two vertical canals enter the posterior wall. From the lower anterior end of the utricle is given off a small membranous tube which passes forward, inward, and downward and unites with a similar tube from the saccule to form the *aquæductus vestibuli*. The aqueduct of the vestibule enters a very small opening on the inner wall of the bony vestibule, and traverses the bone in a curved direction inward and somewhat backward to emerge by a slit-like opening upon the posterior surface of the petrous bone, some 7 or 8 mm. behind the internal auditory meatus. It here expands into a closed sac, the *saccus endolymphaticus*. The endolymph spaces of the labyrinth do not, therefore, communicate directly with the cerebrospinal channels or subarachnoid space. The perilymph of the labyrinth may, on the other hand, escape through the aquæductus cochleæ to mingle directly with the cerebrospinal fluid.

The Saccule.—The other membranous compartment of the vestibule is lodged in the recessus sphericus, a somewhat circular depression on the inner wall of the osseous labyrinth, in front of the crista vestibuli. The saccule is only partly separated by the crista vestibuli from the utricle,

these bodies being in contact above (Schäfer), but having no direct communication. The recessus sphericus, sometimes called the *fovea hemispherica*, and the anterior surface of the crista vestibuli present numerous small perforations through which the saccular branches of the vestibular nerve pass. These nerves penetrate the contiguous surface of the saccule, and there give rise to pronounced structural changes which will be described later. As a result of these localized changes, the cavity of the saccule presents a circumscribed bulging of the inner wall, known as its *macula acustica*. Very similar changes are found in the ampullæ of the three semicircular canals, being there spoken of as the *cristæ acusticæ*. From the lower part of the saccule a minute membranous canal, the *canalis reuniens*, passes downward to enter the scala media of the cochlea just above its closed vestibular extremity. Another small membranous tube leaves the lower posterior aspect of the saccule and passes downward and backward to unite with a similar canal from the utricle. The union of these little canals forms the *aquæductus vestibuli*, already described. The aquæductus vestibuli is also spoken of as the *aqueduct of the endolymph*. The junction of the two canals by which it is formed provides the only communication between the utricle and saccule.

The Cristæ Acusticæ and Maculæ Acusticæ.—The cristæ acusticæ of the canals and maculæ acusticæ of the utricle and saccule are structurally very similar. The crista acustica of each semicircular canal is situated in that part of the membranous ampulla which is attached to the outer wall of the osseous ampulla,—*i.e.*, that aspect of the bony ampulla which is continuous with the outer convex wall of the bony canal. Each crista acustica occurs as a transverse elevation or ridge across its ampulla (Fig. 155). The macula acustica of the utricle is attached to the posterior surface of the crista vestibuli and the recessus ellipticus behind it; that of the saccule is attached to the anterior surface of the crista vestibuli, and in front of this to the recessus sphericus. Both maculæ occur as irregularly round or oval elevations which project from the inner walls into the cavities of the saccule and utricle respectively.

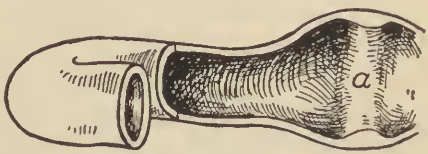

Fig. 155.—Diagrammatic section of ampullar end of horizontal semicircular canal showing position (*a*) of crista acustica.

In the cristæ acusticæ and maculæ acusticæ alike, the following layers may be recognized: 1st, an outer layer of loose fibrous tissue which receives the blood-vessels and branches of the vestibular nerve; this layer is directly connected with the endosteum covering the contiguous bone surface; 2d, a clear or nearly transparent structure known as the tunica propria; and, 3d, the inner layer, consisting of so-called neuro-epithelium, which is made up of the following structures: superficially, a surface layer of elongated pear-shaped, or "flask-shaped," cells, known as the *hair-cells*

(Fig. 156, *a*). They are arranged parallel to each other, their long axes being at right angles to the free epithelial surface. Their lower rounded extremities do not extend downward as far as to the tunica propria. From the upper extremities of these cells, hair-like processes project into the

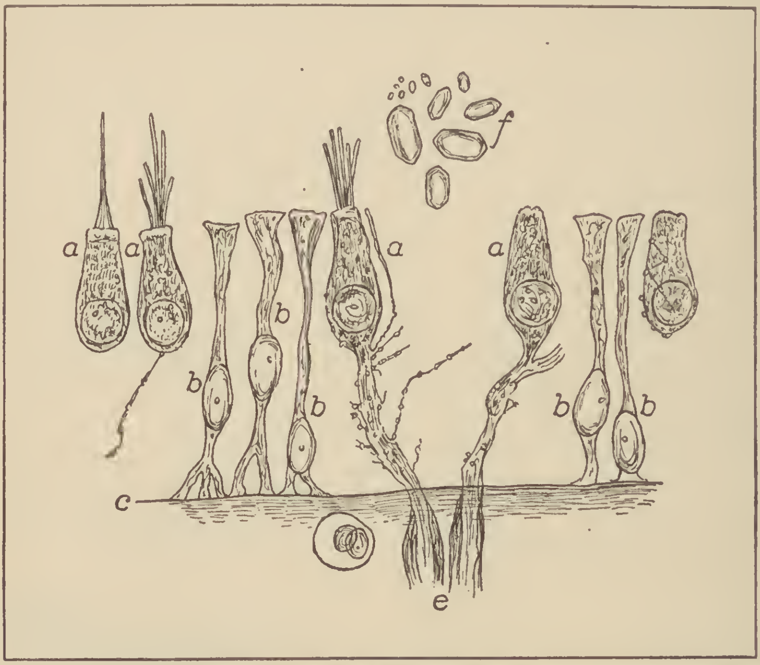

FIG. 156.—Structures common to the cristæ acusticæ and maculæ acusticæ (after Retzius). *a*, Hair-cells; *b*, supporting cells of Retzius; *c*, outer fibrous layer; *e*, nerve-fibres which, before leaving the fibrous layer, lose their medullary sheaths; *f*, otoliths.

cavity of the organ of which they form a part,—*i.e.*, ampulla, utricle, or saccule. In the cristæ acusticæ of the ampullæ, the processes are covered by a gelatinous substance which is spoken of as the *cupola*. In the maculæ acusticæ of the utricle and saccule, they project into the gelatinous covering in which are contained the small bodies known as otoliths. Between the hair-cells and the tunica propria, and in contact with the latter, are elongated nucleated cells known as the fibre-cells of Retzius (Fig. 156, *b*). They are regarded as supporting structures, analogous in this respect to Deiters's cells in the organ of Corti, the sensory function being credited solely to the hair-cells.

The outer fibrous layer is rich in blood-vessels and receives the branches of the vestibular nerve. In it the arteries break up into a network of fine branches. The nerve-fibres, as they leave the fibrous layer, lose their medullary sheaths, the axis-cylinders being continued upward into the epithelial layer, where their branches pass between and in contact with

the cells. They have not, however, been traced into the hair-cells, and the exact distribution of their terminal filaments has not yet been determined.

The structural arrangement above described characterizes both the cristæ acusticæ of the ampullæ and the maculæ acusticæ of the saccule and utricle. In the gelatinous substance covering the latter, and in contact with the hair-cells, clusters of minute bodies, known as *otoliths*, are always present. They consist of crystals of carbonate of calcium. Otoliths are not regularly found in the cristæ acusticæ of the ampullæ, though their occasional presence in small numbers has been observed (Schäfer).

The Membranous Cochlea.—It will be remembered that the spiral tube of the cochlea is divided by the spiral lamina and basilar membrane into two channels,—viz., an inner tube, the *scala tympani;* and an outer, known as the *scala vestibuli*. There remains to be described a third channel, the *scala media*, which contains the organ *of Corti*, the essential structure in the organ of hearing.

The spiral lamina (Fig. 157, *j*) is composed of two bony plates between which are channels transmitting the branches of the cochlear nerve. In

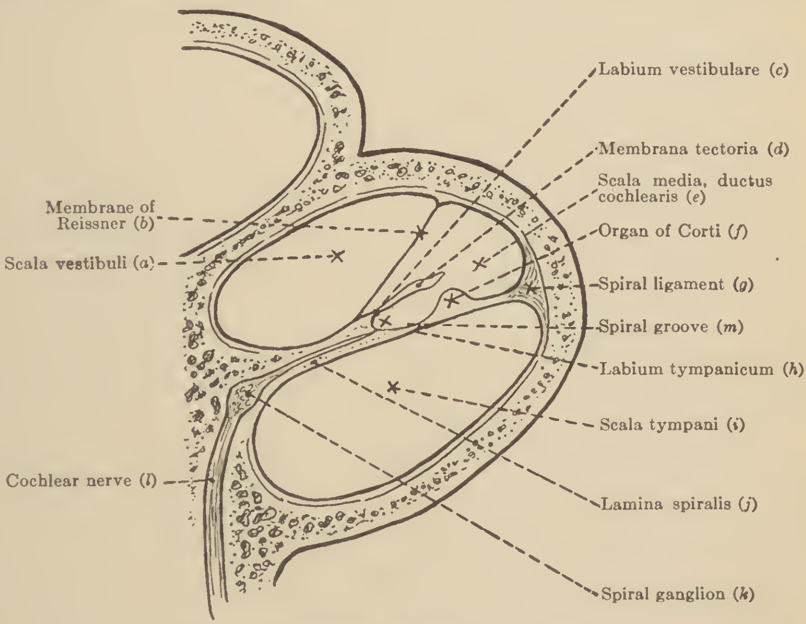

Fig. 157.—Diagrammatic cross-section of cochlear canal.

the dry or macerated bone, the outer edge of the lamina spiralis presents simply a rather sharp edge. In the recent state, however, the outer part is covered with a rather thick layer of connective tissue, and presents on section a grooved formation, described as suggesting the letter C. The lower margin of this groove, which extends further outward toward the outer wall of the spiral tube than the upper, gives attachment to the basilar

membrane. It is called the *labium tympanicum* (*h*). The upper margin is called the *labium vestibulare* (*c*), and the groove between the two, the *spiral groove* (*m*). The labium vestibulare, with the thickened surface immediately behind it, is called the *limbus*. The limbus is formed of firm connective tissue, which, however, disappears after maceration. The limbus gives attachment to the *membrana tectoria* (*d*). From the upper surface of the lamina spiralis, a little behind the attachment of the membrana tectoria, a delicate connective-tissue membrane passes obliquely outward to the outer wall of the spiral tube. This is known as Reissner's membrane (*b*). It encloses a third channel, triangular in form, and situated in the outer part of the cochlear tube between the scala vestibuli and the scala tympani. This is called the *scala media*, *canal of the cochlea*, or *ductus cochlearis* (*e*). The ductus cochlearis runs through the spiral tube from the cochlear base to its apex, and encloses the *organ of Corti* (*f*), to be described later. Its apical extremity ends in a blind sac which is attached to the cupola. The basal or vestibular end also presents a closed extremity, though it receives a little above this a small membranous canal from the saccule, the *canalis reuniens*. The ductus cochlearis contains the endolymph of the cochlea, as distinguished from the scala vestibuli and scala tympani which contain perilymph.

The spiral membrane separates the cochlear duct, or canal, from the scala tympani. Along the line of its attachment to the outer wall of the spiral tube of the cochlea, the lining membrane of the latter is greatly thickened so as to present on section a triangular surface. This spiral projection or ridge is known as the spiral ligament (*g*). Structurally the spiral membrane consists of a homogeneous substance in which innumerable radiating fibres (about 24,000 in all, Retzius) are stretched from the free edge of the spiral lamina to the spiral ligament. It is narrowest at the basal or vestibular end, from which point it becomes gradually wider, reaching its greatest width near the helicotrema. Within the cochlear duct (scala media) and resting upon the basilar membrane is the organ of Corti (*f*).

The Organ of Corti (Fig. 158) occurs as a rather broad elevation or ridge resting upon the basilar membrane throughout the entire length of the cochlear duct. On cross-section, and under high magnifying power, the following structures are observed: Resting directly upon the spiral membrane and near its attachment to the lamina spiralis are two striated rod-shaped structures, known as the *rods of Corti* (I.R.C., O.R.C). That nearest the limbus is called the inner rod of Corti, and the one external to this, the outer rod. Below, as they rest upon the spiral membrane, there is a distinct space between them, but above they incline toward each other so as to meet at their upper extremities. The inner rod is described as resembling the human ulna in form, presenting an upper extremity somewhat similar to the head of that bone. The upper end of the outer rod is compared to the head of a swan,—the back of the head representing the part which fits into the concavity on the upper end of the inner rod,

and the projecting part, resembling the swan's bill, being directed backward (Fig. 159). The rods, both inner and outer, are arranged in single parallel rows extending throughout the entire length of the organ of Corti, and the inner and outer rows enclose between them a triangular space, known as *Corti's tunnel* (Fig. 158, T. C.). Covering the organ of Corti on both sides of the rods of Corti are the characteristic cell-bodies known as the hair-cells (I. H. C., O. H. C.). These are somewhat columnar-shaped cells, narrow above, expanded and rounded below. From their upper extremities tufts of hair-processes project. There is a single row of hair-cells internal to the inner rod of Corti and four rows external to the outer rod. These are known as the *inner hair-cells* and *outer hair-cells* respectively. Beneath the hair-cells, and between them and the basilar membrane, are a number of supporting cells of very different type, known as *Deiters's cells*. These cells are cylindrical below and rest directly on the basilar membrane. Above they become narrowed, each into a slender process which extends upward between the hair-cells, there expanding into a thickened extremity, known as the *pharyngeal process*. These processes of Deiters's cells with their terminal enlargements, or phalanges, enter into the formation of a reticulum surrounding the hair-cells and presenting spaces above through which the hair-processes project. External to the hair-cells and supporting cells of Deiters are several rows of elongated cells which form the outer edge of Corti's organ. They are known as *Hensen's cells* (H. C.). External to this, the basilar membrane is covered by a single row of cubical cells, called the *cells of Claudius* (Cl. C.).

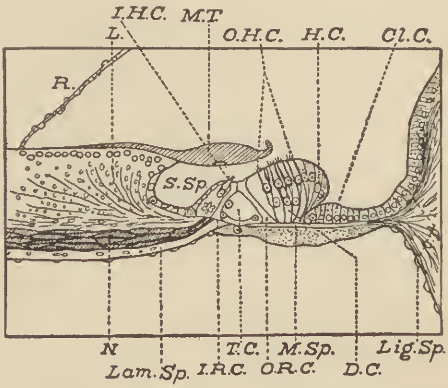

Fig. 158. — Organ of Corti (after Schäfer). *R*, Membrane of Reissner; *L*, limbus; *N*, cochlear nerve; *Lam. sp.*, lamina spiralis; *I. H. C.*, inner hair-cells; *O. H. C.*, outer hair-cells; *I. R. C.*, inner rod of Corti; *O. R. C.*, outer rod of Corti; *T. C.*, tunnel of Corti; *M. sp.*, membrana spiralis; *D. C.*, cells of Deiters; *H. C.*, Hensen's cells; *Cl. c.*, cells of Claudius; *Lig. sp.*, ligamentum spirale.

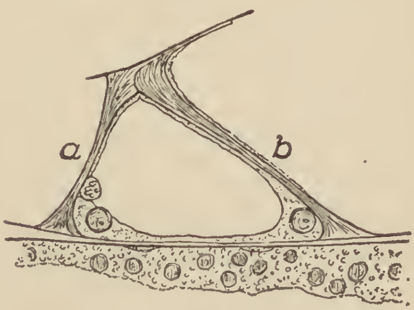

Fig. 159.—Inner (*a*) and outer (*b*) rods of Corti (after Retzius).

The membrana tectoria (Fig. 158, M.T.) is a peculiar structure described by Schäfer as being composed of "fine fibrils embedded in a gelatinous matrix." It is attached by a thin edge to the limbus external to the origin

of Reissner's membrane. From this point it extends outward over the organ of Corti. From its attachment to the limbus to its outer extremity, it increases greatly in thickness. Its upper surface is somewhat convex; its under surface—*i.e.*, that apposed to, or covering, Corti's organ —is irregularly concave. Owing to its varying appearances in different preparations, its dimensions are variously described by different authors. Thus Schäfer states that it extends externally only over the region of the outer hair-cells, while A. A. Gray states that it extends outward as far as the extreme outer limit of the organ of Corti. As seen in microscopic sections a very considerable space usually intervenes between the free surface of Corti's organ and the superimposed tectorial membrane. Apparently most observers believe, however, that the normal relation of the two structures during life is one of actual contact (Schäfer, A. A. Gray, Shambaugh).

The Cochlear Nerve.—The cochlear branches of the auditory nerve enter the cochlea by numerous minute perforations in the circular depression at the base of the modiolus (fossa cochlearis, Fig. 145, *a*). This depression is situated in the anterior wall of the internal auditory meatus, near its fundus, and below the opening of the facial canal from which it is separated by a prominent ridge. It presents a central orifice leading into the central canal of the modiolus, around which are ranged the minute perforations, or foramina (tractus spiralis foraminosus), through which pass the branches supplying the first two turns of the cochlea. The nerve-fibres entering this tract pass directly to the base of the spiral lamina. At the base of the spiral lamina as it winds around the modiolus is a spiral space in which are collected ganglion-cells, forming the spiral ganglion of the cochlea (Fig. 157, *k*). From the cells of this ganglion nerve-fibres radiate outward in the channels between the plates of the spiral lamina, perforating its outer plate to reach the basilar membrane and organ of Corti. On entering the neuro-epithelium of Corti's organ, the nerve-filaments lose their medullary sheaths, their axis-cylinders then passing between, and in contact with, the supporting cells of Deiters, the rods of Corti, and the hair-cells. They have not, however, been traced directly into the hair-cells, and their exact terminal distribution is not yet known.

The nerve-fibres passing through the central canal of the modiolus are distributed to the last half-turn of the cochlea.

Vestibular Nerve.—The distribution of the peripheral branches of the vestibular nerve has already been described in connection with the cristæ acusticæ and maculæ acusticæ of the vestibular apparatus. In the internal auditory meatus the vestibular portion of the auditory nerve is divided into two branches,—*i.e.*, an upper branch which supplies the utricle and ampullæ of the horizontal and anterior vertical canals, and a lower branch which supplies the saccule and ampulla of the posterior vertical canal.

Central Fibres.—Within the internal auditory meatus the cochlear and vestibular branches are traced to their common trunk, which passes in-

ward across the subarachnoid space toward the restiform body. According to Dana, the nerve enters the medulla by two roots,—viz., a lateral or posterior root, composed chiefly of auditory, or acoustic, fibres, and a median or anterior root, made up chiefly of vestibular fibres. These roots communicate with three nuclei,—(1) the central nucleus (acoustic tubercle), situated on the floor of the fourth ventricle; (2) the ventral or accessory nucleus, which springs from the lateral root and lies between it and the median root; and (3) the large-celled nucleus (Deiters's nucleus), which lies external to and below the central nucleus. The lateral root (auditory) communicates chiefly with the accessory nucleus, but is connected also with the other nuclei. From the central and accessory nuclei auditory fibres are sent to the temporal lobes of both hemispheres, but more fibres go to the opposite than to the corresponding side of the brain. The cortical centre for hearing is located in the first and second convolutions of the temporal lobe; and, since the basal nuclei of either ear send more auditory fibres to the opposite side of the brain than its own, it is clear that destruction of the cortical centre on one side will result in impairment of function which will be more pronounced in the ear opposite to the cortical lesion.

The median root of the auditory nerve, composed chiefly of vestibular fibres, is principally connected with Deiters's nucleus and, through fibres leading from Deiters's nucleus, with the cerebellum.

The Blood-vessels.—The arteries supplying the labyrinth are derived from one vessel,—the internal auditory artery, a branch of the basilar artery. This vessel breaks up within the internal auditory meatus into branches which in a general way follow the course of the branches of the auditory nerve. To the vestibule it supplies three branches,—one to the utricle and the horizontal and anterior vertical canals, another to the posterior vertical canal, and a third to the saccule. The cochlear division, before entering the spaces in the spiral lamina, breaks up into a network of anastomotic loops, from which small terminal vessels are sent to supply small circumscribed areas of the basilar membrane, organ of Corti, and outer wall of the cochlear duct (Shambaugh). According to Siebenmann, the venous blood is returned mainly along three channels,—viz., veins leading from the cochlea into the internal auditory meatus, and veins leaving the labyrinth by the aquæductus vestibuli and aquæductus cochleæ respectively. Shambaugh,[1] who made careful studies of the blood-vessels of the labyrinth in the pig, sheep, and calf, found in these animals but one venous channel, which left the labyrinth along the aquæductus cochleæ and apparently drained the entire labyrinth. He refers to the work of Eichler upon the blood-vessels of the human ear, who "found but two routes by which the venous blood left the labyrinth, the vein of the aquæductus cochleæ and the vein of the aquæductus vestibuli." Eichler's conclusions have received the support of Politzer.

[1] Shambaugh: Some Relations of the Blood Supply of the Inner Ear, Arch. of Otol., vol. xxxv, No. 1, 1906.

PHYSIOLOGY OF SOUND PERCEPTION (COCHLEAR FUNCTION).—In attempting to place before the reader a brief synopsis of this rather difficult subject, it may be well to acknowledge that the modern conception of the physiology of sound perception is based largely upon hypothesis and only to a limited extent upon demonstrable facts. There are, however, a few basic facts relating to the cochlear function, acceptance of which may now be assumed. We know, for example, that the cochlea is supplied by the cochlear branch of the auditory nerve, and that, when this cochlear nerve is completely destroyed or divided, total deafness of the corresponding ear results. We know also that destruction of the cochlea, either by disease or by surgical removal, invariably gives rise to total deafness. Of this there can be no shadow of doubt. The occasional reports of cases in which there has been apparent retention of hearing after surgical removal of the cochlea are unquestionably to be explained by the failure of the observer to exclude the compensatory function of the sound ear.

Since, therefore, we may produce deafness by destroying either the cochlea itself or the cochlear nerve before it enters the cochlear fossa at the base of the modiolus, and since the branches of the cochlear nerve have been traced to the basilar membrane and organ of Corti, we may confidently assume that the basilar membrane and organ of Corti are structures which are essential to the function of tone perception. So far we are dealing with indisputable fact. Attempting to go beyond this, we come quickly upon Helmholtz's theory of tone analysis, a hypothesis which has formed the basis of most subsequent investigations which have yielded practical results.

Up to the time of Helmholtz's investigations, the study of the physiology of the cochlea had been obscured by certain mistaken views as to the anatomy of the labyrinth. In the seventeenth and eighteenth centuries it was generally believed that the labyrinth spaces contained air instead of fluid; and later the conception of the cochlear function was distorted by the belief that the fibres of the basilar membrane were longest at the cochlear base and diminished gradually as the apex was approached. We now know that exactly the reverse is true.

Helmholtz's theory is to the effect that the perception of musical tones is brought about by vibration of the basilar membrane in response to sound waves from without, and that the analysis of musical sound into tones of different pitch is explained by the hypothesis that different parts of the basilar membrane vibrate in response to sound waves of different pitch. In other words, that a sound wave consisting of a certain number of rhythmic double vibrations per second can induce movements only in certain fibres of the basilar membrane, other fibres being stimulated only when the number of rhythmic vibrations is either increased or diminished. And since the radiating fibres of the basilar membrane—*i.e.*, stretching from the outer edge of the spiral lamina to the spiral ligament—are longest at the apex of the cochlear pyramid and shortest at the tympanic-vestibular end, it is assumed that the highest tones of the musical scale are produced

by movements of that portion of the basilar membrane nearest the vestibular and round windows, and that the lowest tones correspond to vibrations of portions of the membrane nearest the apex or helicotrema.

The Helmoltz theory is the theory of sympathetic vibration; *i.e.*, it proclaims that only certain parts, or certain fibres of the basilar membrane can respond to, or vibrate in response to, an ærial stimulus, or vibration, of given pitch, or vibration rate. At best it is a hypothesis, but a hypothesis which threw enormous light upon the significance of the structurally complex mechanism of the organ of Corti. The objections to it have been chiefly upon physical grounds; *e.g.*, the unsuitable conditions for vibration, as we understand it, inherent in, and surrounding, the radiating fibres of the basilar membrane. This, it seems to the writer, is in accordance with a näive and common human propensity, which insists upon literal translation of terms, and accepts with difficulty any thesis which cannot be explained by analogy with commonly known facts and phenomena. Admittedly the radiating fibres of the basilar membrane are not analogous to the strings of a piano in their mode of sympathetic response to sonorous stimuli from without.

That certain definite areas of the basilar membrane and organ of Corti have to do with the perception of certain tones has been supported by the studies of Wittmaack.[2] Wittmaack's experiments consisted in subjecting animals during prolonged periods to hearing constantly a certain note,—*e.g.*, 256 d.v., 1024 d.v., etc.,—the animals later being killed and the cochlear structures examined under the microscope. It was found that certain definite areas of Corti's organ, corresponding constantly to certain tones, gave evidence of pathologic change as a result of the prolonged stimulation or strain; and, further, that high tones produced morbid changes near the vestibular end of the scala media, and that low tones gave rise to changes nearer the helicotrema. These results have been confirmed by the investigations of Siebenmann.

A belief which has become part of the accepted hypothesis is to the effect that some interaction between the organ of Corti and the membrana tectoria is essential to the proper transmission of auditory impressions to the brain,—or, to be more explicit, that friction or impact of the projecting hair-processes of the hair-cells covering the organ of Corti against the under surface of the tectorial membrane is in some way essential to the function of tone perception. As to the method, or process, by which this contact or friction is brought about, the consensus of opinion among physiologists has strongly supported the view that sound waves, propagated through the perilymph and reaching the under surface of the basilar membrane, have there caused vibration or displacement of certain of its fibres thus carrying the corresponding hair-processes of Corti's organ against the tectorial membrane.

Naturally many theories and modifications of theories of sound per-

[2] Wittmaack: Ueber Schaedigung des Gehör durch Schalleinwirtung, Zeitsch. f. Ohrenheil., Bd. 50, 1908.

ception have from time to time been advanced which, though exceedingly interesting, cannot be brought within the limited space of this handbook.

The theory of Sir Thomas Wrightson and Sir Arthur Keith is based upon an intensive study of wave forms and elaborate experiments to determine the direction of fluid sound waves within the cochlea and to explain mechanically the influence thereby exerted upon the essential structures of the organ of Corti. It is assumed that each inward movement of the stapedial foot plate imparts to the cochlear fluids an impulse which is immediately transmitted throughout their entire mass. According to this theory, the fluid sound wave travels, not by way of the scala vestubuli and through the helicotrema to the scala tympani, but passes directly through Reissner's membrana and the scala media to the scala tympani, this mass movement being, of course, compensated for by an outward movement of the membrane of the round window. With these mass movements of the cochlear fluids the basilar membrane and the organ of Corti are depressed inward toward the scala tympani. The interaction of the hair-cells of Corti's organ and the membrana tectoria are brought about by the to and fro movements of the basilar membrane. An effort is made to bring the movements of the hairs of the hair-cells into relation with what Wrightson calls the four phases of a simple sound wave, or rather the four phases of a single double vibration.

Wrightson's theory apparently relegates Reissner's membrane and in fact the scala media to a status of complete functional insignificance. Though ingenious and interesting, it is, in the author's opinion, unconvincing.

The theories of Siebenmann and Shambaugh, giving a major function in vibration to the membrana tectoria, have found comparatively few followers, one obstacle to their acceptance being the apparently inherent unfitness of this structure to assume this rôle.

Of modifications of the Helmholtz theory, the most satisfying, in the author's opinion, is that of Dr. Albert A. Gray of Glasgow. According to this theory, the basilar membrane may vibrate in response to a sound wave over a considerable area; but there is a point of maximum amplitude of vibration, varying with the pitch or vibration rate. In other words, while the vibration may involve considerable portions of the basilar membrane, the point of maximum amplitude varies with each change of pitch, or vibration rate; and it is the note corresponding to this point of maximum vibration which is recorded in the cerebral hearing centre, and therefore appreciated as a musical tone. This theory is the more satisfying for the reason that, while robbing the Helmholtz theory of none of its essential features, it removes one of the chief obstacles to its acceptance, *i.e.*, the conception of vibrations propagated through the cochlear perilymph which would not

[3] Siebenmann: Bardelben's Handb. d. Anat. d. Menschen. 1897.

[4] Shambaugh: Physiology of the Cochlea, Annals of Otol., Sept., 1910; Physiology of Tone Perception, Dec., 1910.

[5] Gray: Various Theories of Hearing; Journal of Laryngol. and Otol., p. 391.

induce movements in the basilar membrane more extensive than are allowed for in the Helmholtz hypothesis.

The Vestibular Apparatus.—The membranous vestibular apparatus consists of the utricle, saccule, and three semicircular canals. Whatever may be their exact function in health, it is probable that these structures act in concert. Injury to any one canal causes subjective and objective phenomena very similar to those following injury of the other two, or to the parts resting within the bony vestibule. The phenomena of vestibular irritation have been carefully studied both experimentally and clinically by Flourens, Goltz, Breuer, Crum-Brown, Ewald, Barany, Neumann, Ruttin, Hinsberg, Jansen, and a host of observers in different parts of the world. These observations, while of immense value in their bearing upon the symptoms of acute tympanic disease, have thrown but little direct light upon the vestibular function in health. They will not, therefore, be considered in the present chapter.

We know that the vestibular mechanism is not an organ essential to man's power of equilibration, for after its complete removal the individual's equilibrium is soon re-established. By a somewhat similar course of logic the theory of Ewald—*i.e.*, that the vestibular apparatus is in some degree responsible for the tone of the skeletal muscles—must be discarded, for destruction of the canals does not seem to influence muscular tone injuriously. The author has had the opportunity of examining two individuals, a man and a woman, in whom the function of both labyrinths was absolutely and apparently permanently ablated, and these patients seemed to be rather above the average in muscular strength and potential efficiency.

It is probable that the vestibular mechanisms are in one sense organs of orientation, in that they enable man under all conditions,—*i.e.*, in light or darkness, and in whatever position his body may be placed,—subconsciously and without effort to determine the position of the different parts of his body. It is the sudden withdrawal of this power which places the individual after removal of one or both labyrinths, and even after he has recovered from the first vestibular disturbances incident to the operation, in some danger of serious accident. That he soon learns to guard against such mishaps under all or any conditions does not disprove the value of these organs in health. It proves simply that certain other faculties—*e.g.*, the so-called muscle, arthrodial, and tactile senses and the sense of sight—have so enlarged their scope as to compensate for that which he has lost. Viewed in this way, it would seem that, if we permitted outselves to speak of the cochlear branch of the auditory nerve as the auditory branch, we might with equal propriety regard the vestibular branch as *the nerve of orientation.*

The Otolith Apparatus.—In recent years there have been recorded several observations of a peculiar form of nystagmus, somewhat resembling, yet clearly distinguishable from, that induced by rotation. Barany [6]

[6] Barany: Disease of the Otolith Apparatus; Journal of Laryng. and Otol., vol. XXXVI, p. 229.

recorded the case of a man suffering from multiple sclerosis who, if the head was inclined toward the right shoulder, no matter how slowly, regularly exhibited a marked horizontal nystagmus to the left. That it was not due to displacement of endolymph in the semi-circular canals, but rather to the effect of position, was proved by the very gradual changes of position which caused it and also by its long persistence, *i.e.*, provided that the inclined position of the head was maintained. This phenomenon is believed to be brought about through a disturbance of the so-called otolith apparatus or, to be more exact, of the saccule and utricle, though the exact nature of the imbalance by which it is produced has not been determined.

During the past few years, much experimental work has been done chiefly in the University of Utrecht and under the direction of Professor Magnus and Dr. deKleijn, to determine more definitely the exact function of the Saccule and Utricle. While these experiments are interesting, their practical bearing upon health and disease has not yet been sufficiently established to call for their inclusion in a handbook on otology. Students who wish to follow these investigations in detail will find in the short and interesting monograph of Dr. Alex. H. Tweedie[7] of Nottingham, England, a brief outline of this work, to which has been appended a rather comprehensive bibliography.

Whatever the exact function of the vestibular mechanism may be, it is fairly certain that the essential structures are the cristæ acusticæ of the semicircular canals and maculæ acusticæ of the saccule and utricle. Both histologically and physiologically there appears to be a certain analogy between these structures and Corti's organ. Thus, both the organ of Corti and the cristæ and maculæ acusticæ are covered by a highly-organized neuro-epithelium, of which the surface strata are composed of hair-cells from which hair-processes project. In each of these organs the hair-processes project into, or toward, an important superimposed structure, friction or impact against which is essential to its proper performance of function. Thus the hair-cells of the cristæ acusticæ do not project directly into the endolymph of the ampullæ, but into the soft *cupola terminalis* covering them. The hair-cells of the maculæ acusticæ are in contact with the otolith-holding membrane, while the hair-cells of Corti's organ project toward the under surface of the membrana tectoria. According to Shambaugh, the relation of the free surface of the organ of Corti and the tectorial membrane is one of actual contact. In the organ of Corti, impact of the hair-processes against the membrana tectoria is brought about through the agency of sound waves propagated through the labyrinthine fluids. In the case of the cristæ acousticæ and maculæ acusticæ, interaction is brought about between their hair-processes and the cupola terminalis and the otolith membrane by sudden changes in the position of the head. The importance of this structural and physiological analogy between the different parts of the membranous labyrinth has been emphasized by Shambaugh.

[7] Tweedie: Saccular, Utricular and Allied Reflexes; Journal of Laryn. and Otol., 1922, p. 213

CHAPTER XI.

INFLAMMATORY AND SUPPURATIVE LESIONS OF THE LABYRINTH.

Suppurative labyrinthitis is unquestionably one of the most dangerous lesions with which the physician has to contend. The disease is practically always secondary to purulent disease of the middle ear or mastoid. The initial symptoms—*i.e.*, those which announce the actual invasion of the membranous labyrinth—are invariably such as may be attributed to vestibular irritation. Unfortunately, universal familiarity with the phenomena of vestibular irritation can not as yet be assumed. It may be well, therefore, before discussing the clinical aspects of the disease, to review briefly certain experiments, facts, and accepted theories upon which our present knowledge of suppurative labyrinthitis depends.

The experiments of Flourens[1] upon pigeons and rabbits, published in 1824, established the fact that section of any one of the semicircular canals gives rise to uncontrollable movements of the head and eyes in the plane of the canal experimented upon. Breuer in 1888 demonstrated that similar phenomena could be induced by simple irritation of a canal without causing its actual destruction,—*e.g.*, by electrical, thermal, or chemical irritants.

In 1892, Professor Ewald, of Strasburg, carried these experiments further, corroborating the results of Flourens, Breuer, and others, and establishing further the relation between endolymph movements in the different canals and the character and direction of the resulting nystagmus. These latter experiments are so clearly the basis of our present knowledge of the phenomena of vestibular irritation that we shall take time to describe them briefly.

Fig. 160.—Semicircular canals of pigeon (after Ewald).

Ewald's Experiments (Fig. 160).—Ewald[2] experimented upon pigeons in the following way: Having exposed the canal selected for investigation, a small hole was drilled into it near its small end, and the lumen of the canal was obliterated at this point by the introduction of a lead mass. A second opening was next made between the small occluded end and

[1] Flourens: Les propriétés et les fonctions du système nerveux, pp. 454–482.
[2] Ewald: Physiologische Untersuchengen über das Endorgon des Nervus Octavus, pp. 255–266.

the large, or ampullar, end of the canal. Into this second opening was introduced and fixed one end of a small hollow cylinder, open at both ends, the outer end of which communicated by means of a piece of narrow rubber tubing with a compressible rubber ball or bulb. The interior of bulb, tubing, and cylinder being now directly continuous with the lumen of the canal, it is clear that compression of the bulb must necessarily cause displacement of the endolymph in the membranous canal involved; and, since the small end of the canal has been obliterated, it is obvious that during compression, the endolymph movement must be toward and through the ampulla and into the cavity of the utricle. Beginning with a partially compressed bulb, it is equally clear that release of pressure will cause a movement of endolymph from the utricle toward and through the ampulla and in the direction of the small end of the canal.

Ewald by this method experimented separately on the three semicircular canals, and obtained the following results:

Right Horizontal Canal.—Compression of the bulb (causing endolymph displacement toward the ampulla) was invariably followed by a slow strong movement of the head, exactly in the plane of the canal, toward the left. Coincidently with this head movement, the eyes were moved, also in the plane of the canal, to the left. On release of pressure the head and eyes quickly returned to their normal position. Suction (causing endolymph movement from the utricle through the ampulla and toward the small end of the canal) was followed by slow turning of the head and eyes, always in the plane of the canal, to the right.

This experiment upon the *left horizontal canal* gives rise to similar movements of the head and eyes, but in reversed directions.

Right Posterior Vertical Canal.—Compression (*i.e.*, endolymph displacement toward and through ampulla) is followed by gradual movement of the head and eyes exactly in the plane of this canal and in the direction of its ampulla,—*i.e.*, to the right. Suction gave rise to movements in the same plane, but in the opposite direction,—*i.e.*, to the left.

The reactions of the *anterior vertical canal* are similar to those of the posterior vertical, varying only in accordance with its different plane.

Ewald's experiments established definitely the following important facts: (1) Excitation of any single canal can produce nystagmus only in a plane parallel with the plane of that canal; (2) the relation between the direction of the endolymph movement in any canal and the direction of the resulting nystagmus is definite and constant; and, consequently, (3) by reversal of the endolymph movement in any canal we can reverse the direction of the induced nystagmus.

The analogy between these experimental phenomena in the lower animals and vestibular nystagmus in man is now clearly established. As we shall see later, vestibular nystagmus in man is characterized by a quick movement in one direction and a slow movement in the opposite direction. With pigeons both head and eyes move in response to vestibular irritation, whereas in man the nystagmic movements are confined to the eyes. With

regard to Ewald's experiments, it is important that we bear in mind the fact that the slow movements of head and eyes caused by either compression or suction correspond to the slow component of a vestibular nystagmus. We are accustomed to name the direction of a vestibular nystagmus in accordance with the direction of the quick eye movement,—*e.g.*, nystagmus to the right, or nystagmus to the left. The quick eye movement does not, however, represent the vestibular impulse, which is responsible only for the slow eye movement in the opposite direction, the quick recoil movement being under the control of the central nervous system.

Note.—The differentiation of the vestibular from the cerebral component of a vestibular nystagmus has been beautifully demonstrated in certain cases in which the caloric experiment—next to be described—has been employed upon a patient under deep anæsthesia. Narcosis having been pushed to the point where the peripheral reflexes are abolished, the caloric test has been applied in the manner usually giving rise to a vestibular nystagmus toward the opposite ear,—let us say to the left. Under these conditions, the vestibular activity being retained and the central nervous control having been suspended, the eyes, instead of executing successive quick movements to the left, are slowly rotated to the right and remain in that position. As the influence of the anæsthetic wears off and the central reflexes are restored, the eyes are suddenly jerked from their position of deviation to the right, and execute the characteristic quick movements to the left. Or, if the narcosis is prolonged, the influence of the vestibular impulse may wear itself out, and the eyes return to their normal position.

Barany's Experiments; the Caloric Reactions.—It has long been known to otologists that irrigation of the ears for the removal of pus or cerumen will in some cases give rise to nystagmus, vertigo, and disturbance of equilibrium. This remained an unsystematized fact of no practical value in otology until the discovery by Barany, of Upsala, that these phenomena were not haphazard occurrences, depending upon individual idiosyncrasy, but were constant reactions having a definite relation to the temperature of the water used.

The caloric reaction may be briefly stated as follows: If we irrigate either ear of a person with normal labyrinths with water of body temperature, no subjective or objective symptoms are experienced. If we use water considerably below blood heat,—*i.e.*, 86° F., or lower,—we invariably obtain the following reactions,—viz.: (a) rotary nystagmus of which the quick movement is in the direction away from the ear irrigated; (b) the patient experiences subjective vertigo, and (c) exhibits marked disturbances of equilibrium. Substituting hot water,—*i.e.*, at 110° F.,—we obtain exactly the same phenomena with the exception that the direction of the nystagmus is now toward the ear irrigated, and the ataxia shows certain changes in accordance with laws governing its relation to the nystagmus present.

These reactions are very nearly invariable with normal persons. Their diagnostic value depends upon the fact that when the static labyrinth (vestibular apparatus) has been destroyed, either surgically or by disease, the vestibular nerve can no longer respond to stimulation by heat or cold, and the caloric reactions are absent.

NOTE.—Barany's explanation of these phenomena in accordance with Ewald's experiments is interesting and on the whole convincing. We must regard the whole labyrinthine cavity as an irregularly shaped vessel containing fluid (perilymph, endolymph), the temperature of which is presumably that of the blood. If, now, we bring hot or cold water in contact with one wall of this vessel, the temperature of that part of the contained fluid nearest this wall will be raised or lowered. In other words, its specific gravity here will be increased or diminished, and it will sink or rise according to the physical laws governing fluids of different specific weight. Now, the parts of the semicircular canal system nearest the surface of the inner tympanic wall are the anterior half of the horizontal canal and the anterior or outer third and ampulla of the anterior vertical canal (Fig. 161). These parts are, therefore, first influenced by the hot or cold water used in irrigating the ear. With the head erect, sudden cooling of the endolymph in the external (horizontal) semicircular canal does not give rise to an endolymph movement, this being prevented by its horizontal position. In the case of the anterior vertical canal, on the other hand, the part nearest the tympanum—viz., the outer or ampullar end—points directly downward. Irrigation with cold water would, therefore, in this canal give rise to a downward movement of endolymph toward and through its ampulla. That cold irrigation in normal individuals is always followed by rotary nystagmus toward the opposite side is in exact accordance with Ewald's experiments. Further corroboration of this theory is obtained from the fact that if, immediately after irrigation with cold water, the head is bent downward so that the top of the head is directly toward the floor,—a position in which the endolymph movement would be reversed,—the direction of the nystagmus is also reversed,—*i.e.*, is toward the ear irrigated. Further, if the head, after irrigation with cold, is quickly bent forward to an angle of 90 degrees so that the face looks directly downward, the nystagmus is changed from the rotary to the horizontal type. The explanation of this is not far to seek, for in this position of the head the anterior vertical and posterior vertical canals assume positions somewhere between the vertical and horizontal planes, while the horizontal canals fall quite in the vertical plane. The chief endolymph movement is therefore in the horizontal canal, with resulting horizontal nystagmus. That the use of hot water, which would reduce endolymph specific gravity, should give rise to nystagmus in reverse directions tends further to support Barany's theory as to the causation of these interesting phenomena.

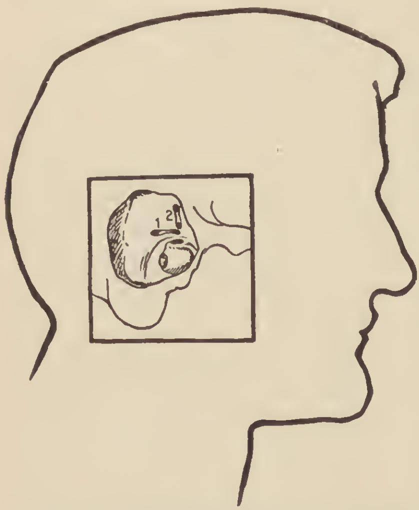

FIG. 161.—Diagrammatic picture of inner tympanic wall, showing (1) horizontal semicircular canal, and (2) anterior end of anterior vertical canal.

VESTIBULAR NYSTAGMUS.—Involuntary eye movements occur as an exceptional phenomenon with several conditions having no relation to disease of the labyrinth,—*e.g.*, cerebellar lesions, certain ocular diseases, hereditary syphilis, neurasthenia. It is necessary, therefore, that a word be said as to certain characteristic features which distinguish vestibular nystagmus from other forms. To establish a claim to vestibular origin,

(1) a nystagmus must be composed of a quick movement in one direction and a slow movement in the opposite direction; (2) it is increased, usually in rapidity and always in length of excursion, when the eyes are turned voluntarily in the direction of the quick movement; (3) it becomes weak, or may disappear wholly, when the eyes are turned in the direction of the slow nystagmic movement.

The above are invariable characteristics of nystagmus of vestibular origin, whether produced by experimental irritation or in the course of acute labyrinthine disease.

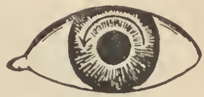

Fig. 162.

Vestibular nystagmus may be horizontal, oblique, vertical, or rotary. The nystagmus caused by acute labyrinthine disease is practically always rotary.

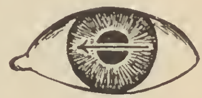

Fig. 163.

Obviously all forms of vestibular nystagmus are rotary, since in all the eye movements take the form of to-and-fro rotation about some imaginary axis. The term "rotary," however, is applied only to forms of nystagmus in which the eye movements as seen from in front do not seem to describe a straight line upon the cornea. We call all types of nystagmus "rotary," therefore, in which our line of vision does not fall in the plane of the nystagmus. Thus, if our line of vision coincides with the axis of rotation,—*i.e.*, is at right angles to the plane of rotation,—we have the most pronounced type of rotary nystagmus possible,—a veritable wheel nystagmus (Fig. 162). On the other hand, when our line of vision falls within the plane of rotation, there results a straight nystagmus which may be horizontal (Fig. 163), vertical (Fig. 164), or oblique (Fig. 165), but can not be rotary. When our line of vision falls somewhere between the axis of rotation and the plane of rotation, we have a form of nystagmus falling somewhere between the straight and the wheel type, and partaking somewhat of the character of each (Fig. 166). In such a nystagmus the excursion made by any given point upon the cornea will, as seen from in front, represent the arc of a circle much larger than one whose radius is measured by the distance between the centre of rotation and the corneal point in question. Such a nystagmus is also called "rotary," and is the type usually seen in acute labyrinthine disease.

Fig. 164.

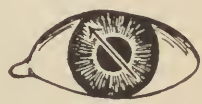

Fig. 165.

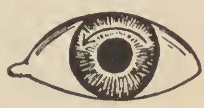

Fig. 166.

Figs. 162-166.—Types of vestibular nystagmus.

From the results of Ewald's experiments, and from a clinical study of nystagmus as seen in suppurative lesions of the labyrinth, we may deduce the following,—viz.: That, since irritation of a single canal can produce nystagmus only in its own plane, and since the nystagmus accompanying acute labyrinthine disease rarely corresponds exactly to the plane of any single canal, we may assume that suppurative invasion of the labyrinth

almost invariably involves all, and always more than one, of the three semicircular canals.

A little practice may be required to detect the rotary character of the nystagmus usually accompanying acute labyrinthine disease. To the unpractised eye the ocular movements may at first seem quite in the horizontal plane. If, however, we fix our attention upon some dilated corneal vessel and note carefully its changing relation to the border of the lower lid, the rotary element becomes unmistakable. Lack of attention to this point has doubtless been responsible for some obviously incorrect clinical reports.

Physiological Nystagmus.—This term has been applied by Barany to a form of nystagmus which is seen in many normal persons when the eyes are voluntarily placed in the extreme lateral position in either direction. It is mentioned in this place because it is of the greatest importance that the student should learn to recognize it as having no significance as an indication of suppurative labyrinthitis. The character of the eye movements is quite similar to that above described,—that is to say, it is usually rotary in character and is composed of a quick movement in one direction and a slow movement in the opposite direction. It is, however, easily differentiated from the nystagmus of labyrinthine disease by the following points:

(a) Spontaneous vestibular nystagmus in its most active stage is constant, but is exaggerated when the eyes are voluntarily turned in the direction of the quick movement. Physiological nystagmus is present only when the eyes are turned strongly in one or the other lateral direction, and then usually lasts but a few seconds.

(b) Vestibular nystagmus in its active stage is present whatever the position of the eyes. Later, however, as the strength of the nystagmus is gradually reduced, it may be wholly absent when the eyes are turned in the direction of the slow nystagmic movement. Physiological nystagmus changes its direction according to the position of the eyes,—the quick movement corresponding to the lateral direction in which the eyes are voluntarily turned.

(c) Vestibular nystagmus in its most active stage is almost invariably accompanied by vertigo and ataxia, and these symptoms, even after the nystagmic movements have grown weaker, can usually be reinduced by sudden movements of the head. Physiological nystagmus is absolutely unaccompanied by any subjective symptoms.

Barany's estimate that this so-called physiological nystagmus occurs in 60 per cent. of normal persons seems to me exaggerated,—20 to 25 per cent. being, according to my observation, nearer the correct proportion.

Rotation or Turning Experiment.—This is simply another method of inducing endolymph movements in the semicircular canals and thereby causing symptoms of vestibular irritation.

When a normal person, seated, with head erect, upon a revolving chair (Fig. 167), is suddenly and forcibly rotated in one or other direction, —let us say to the right,—there occurs a horizontal nystagmus with the

FIG. 167.—Revolving chair: patient and physician in position for rotation test.

quick eye movement in the direction in which he is turned,—*i.e.*, to the right. When the rotations are suddenly stopped, the direction of the nystagmus is reversed,—*i.e.*, it is now to the left. The average duration of this so-called "after nystagmus" is about 40 seconds.

The rotation experiment is a useful aid in the study of vestibular phenomena in that, by placing the head during rotation in various positions, one is able to induce at will almost any form of vestibular nystagmus. Obviously the various forms of nystagmus, vertigo, and ataxia can mean little to the student who has not a correct mental picture of the planes of the three semicircular canals, and also of the relative positions of their ampullar and small ends. These relations seem often to present such difficulties to the beginner that the writer is tempted to suggest a very simple device by which, with the aid of two visiting cards, one may easily fix them in memory.

In Fig. 168, *a–b* represents a line throughout which the card is to be cut or divided by a sharp-pointed knife; *c–d*, a line at which the card is to be bent or folded upon itself. If now we bend the card at *c–d*, so that the

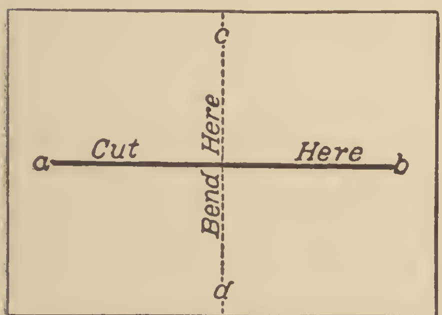

FIG. 168.

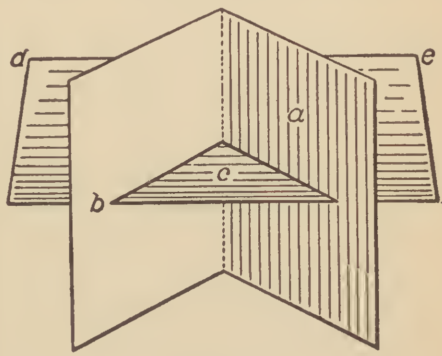

FIG. 169.

FIGS. 168 and 169.—Author's scheme for remembering semicircular canal planes.

two segments occupy planes at right angles to each other, and insert between the cut edges, now also bent at right angles, the edge of a second card (Fig. 169), we have three planes, each at right angles to the other two.

If we wish this little improvised model to represent the planes of the three canals of the right ear, we have only to hold it (mentally) to the side of the head, with the bisecting card in the horizontal plane, and its edge, *d–e*, parallel with the anteroposterior axis of the skull. In this position we have the upper half of the anterior segment of the vertical card (*a*) representing the plane and position of the anterior vertical canal, the middle part of the posterior segment (*b*) representing the posterior vertical canal, and the enclosed part of the inserted card (*c*) representing plane and position of the horizontal canal.

If we wish this model to represent also the respective curves and the

ampullar and small ends of the three canals, we must outline these structures upon the cards as indicated in Figs. 170 and 171, and with scissors cut away part of the card (Fig. 171), as indicated by the dotted lines. When these cards are cut, folded, and adjusted (Fig. 172), we have clearly before us the common opening of the anterior and posterior vertical canals (a), the ampulla of the anterior vertical canal (b), the ampulla of the horizontal canal (c); and the ampulla of the posterior vertical canal (d).

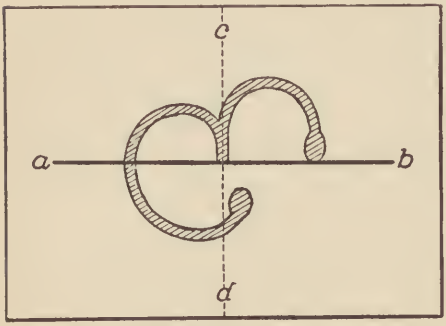

FIG. 170.

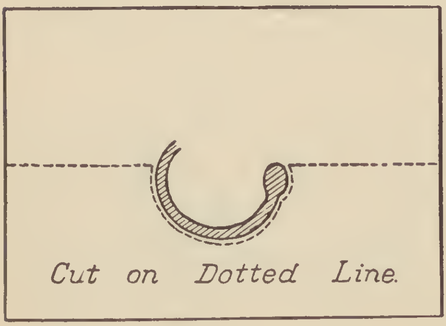

FIG. 171.

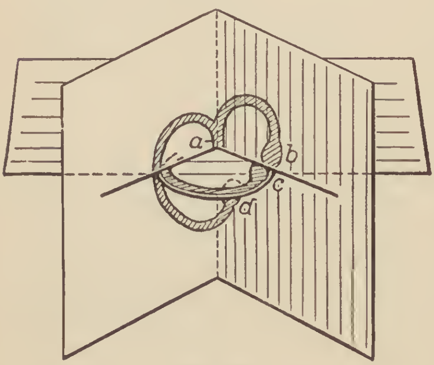

FIG. 172.

FIGS. 170, 171, and 172.—Author's scheme for remembering exact relative positions of the three canals.

Explanation of Rotational Nystagmus.—When the head, held in the erect position, is suddenly turned in the horizontal plane either to the right or left, the endolymph in the two horizontal canals, by reason of its inertia, at first lags behind,—*i.e.*, is displaced in the opposite direction. If, for example, the head is turned to the right, the initial endolymph movement will in the right horizontal canal be toward its ampulla, while in the left horizontal it will be toward the small end of the canal (see Fig. 173). Now, according to Ewald's experiments, these are precisely the endolymph movements which in these canals should produce nystagmus to the right, and this phenomenon is always present during the rotations to the right. When the rotations are suddenly stopped, the endolymph, this time by reason of its momentum, is displaced in the opposite direction, with the result that the direction of the nystagmus is reversed,—*i.e.*, it is now to the left.

NOTE.—When a normal individual is rotated about a vertical axis, the influence of the rotations upon any particular canal will depend upon the relation which the plane of this canal bears to the horizontal plane. If the canal in question lies quite in the

horizontal plane, the influence of rotation will reach its maximum, and will produce a maximum endolymph displacement. As the plane of the canal departs from the horizontal, the influence of rotation in the horizontal plane is diminished, and becomes progressively less in exact proportion as the angle of extension between the plane of the canal and the horizontal plane is increased. Finally, when the canal assumes the vertical plane, no displacement of endolymph results from rotation about a vertical axis. It is obvious, therefore, that with head erect, rotation in the horizontal plane influences only the two horizontal canals, the anterior and posterior vertical canals being eliminated by their position. If, however, the head during rotation is bent forward so that the face looks directly downward, the horizontal canals will be made to assume the vertical plane, while the posterior vertical canals will be brought more into the horizontal plane. Rotation with the head in this position will therefore be followed by rotary nystagmus.

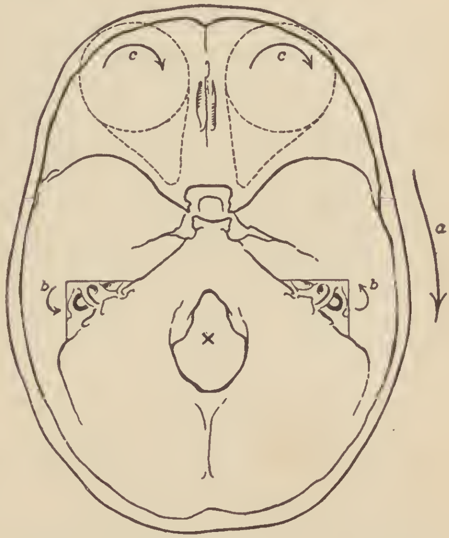

Fig. 173. — Diagrammatic picture of a horizontal section of skull, passing through horizontal semicircular canals. *a*, Large arrow indicating direction in which individual is rotated; *b*, small arrow = resulting endolymph displacement during rotation; *c*, medium arrow showing direction of quick eye movement during rotation.

During rotation, then, we may, by changes in the position of the head, bring different canals under the influence of the experiment, and thus vary at will the form of nystagmus. In all cases, however, the direction of the nystagmus follows a definite rule, which may be briefly restated as follows: *During rotation a person exhibits nystagmus in the direction in which he is turned. Arrest of rotation is immediately followed by nystagmus in the opposite direction.*

The diagnostic value of the rotation test and the method of applying it will be spoken of in connection with the latent stage of suppurative labyrinthitis.

The Symptom Complex of Vestibular Irritation: Nystagmus, Vertigo, Ataxia.—Vestibular irritation, whether experimentally induced (*e.g.*, by thermal, electrical, or mechanical stimuli) or resulting from acute labyrinthine disease, is almost invariably announced by three associated symptoms,—viz., spontaneous nystagmus, vertigo, and disturbance of equilibrium. These three phenomena are so constantly associated with the onset or acute stage of suppurative labyrinthitis that their absence during this period would of itself be sufficient to exclude the vestibule and static labyrinth as among the parts involved.

Obviously, these symptoms, either singly or in association, may also be present in various conditions not dependent upon labyrinthine disease. Thus, nystagmus may occur with certain ocular lesions, with cerebellar abscess, with tumors occupying the posterior fossa of the skull, or may occur

as a physiological anomaly having no recognized pathological significance ("physiological nystagmus"). As with nystagmus, so may vertigo and ataxia result from many functional and organic disorders. It is very important, therefore, that we be able to recognize certain distinguishing features, either in the symptoms themselves or in their relation to each other, as they occur in acute labyrinthine disease.

In the first place, vestibular vertigo is always rotary in character,—*i.e.*, is always accompanied by a subjective impression of the rotation of surrounding objects, and this subjective rotation is always in a plane corresponding to the plane of the nystagmus. There can be no doubt that the very closest relation exists between vestibular vertigo and vestibular ataxia, and that the character of each depends upon the type of nystagmus present. Barany was the first to observe certain seemingly constant relations between vestibular vertigo and ataxia and vestibular nystagmus, which he formulated somewhat as follows:

1. *Spontaneous vertigo of vestibular origin is always accompanied by some degree of spontaneous vestibular nystagmus, and is always increased when the eyes are voluntarily turned in the direction of the quick nystagmic movement.*

2. *Vestibular ataxia is always accompanied by nystagmus, and is always influenced by the position of the head.*

3. *A person exhibiting vestibular nystagmus tends to move within the plane of the nystagmus, and to fall in the direction opposite to the quick nystagmic movement.*

The writer believes that the above statements are in the main correct, and that what may seem to be occasional exceptions or contradictions will under more accurate methods of observation be recognized as apparent rather than real. The practical value of these hypotheses in furnishing us with criteria by which the value of single symptoms may be gauged must be apparent. Thus, vertigo which is not accompanied by nystagmus even when the eyes are turned strongly in one or other lateral direction, and ataxia which is not attended by nystagmus and is not influenced by changes in the position of the head, are certainly not suggestive of vestibular irritation.

A word must also be said as to the *reaction movement*, or tendency which a person exhibiting vestibular nystagmus shows to fall in a certain direction. Barany's law, it will be remembered, reads, "A person exhibiting vestibular nystagmus tends to move within the plane of the nystagmus, and to fall in the direction opposite to the quick nystagmic movement." It seems to me that we shall obtain a better understanding of the principle involved if we say that a person exhibiting vestibular nystagmus *tends to rotate within the plane of the nystagmus and in the direction opposite to the quick eye movement.* This tendency to rotation is about an axis passing through his head, and he falls, or tends to fall, in the direction in which this rotation throws his body, and this, as we shall see, is not always in the opposite direction to the quick nystagmic movements.

Let us take for example a person who has been turned rapidly in a revolving chair ten times *to the right*, and who as a result exhibits well-marked horizontal nystagmus *to the left*. He now experiences pronounced rotary vertigo in which surrounding objects seem to rotate about him in the horizontal plane,—and usually to the left. Immediately after the rotations of the revolving chair are stopped, let him stand and, with head erect and feet approximated, close his eyes. The nystagmus being in the horizontal plane, the reaction movement should be, not falling, but gradual turning in the horizontal plane to the right (Fig. 174). This, however, may not be demonstrated, or may be shown only by a tendency to turn the head to the right. Let us now test Barany's proposition that *vestibular ataxia is always influenced by the position of the head.* Request him to incline the head forward to an angle of 90 degrees so that the face looks directly downward (Fig. 175). With this position of the head the plane of the nystagmus is changed from the horizontal to the vertical, and, the quick eye movement being to the left, the head tends to rotate to the right. This rotation of the head to the right results, however, in throwing his body in the opposite direction, and he falls to the left. In this case we seem at first glance to have a contradictory reaction, in that the subject falls in the direction of the quick eye movement. After a moment's reflection, however, and with Fig. 175 before us, it becomes clear that the rotation of the head in the direction opposite to that of the quick eye movement is precisely the factor which determines his falling to the left. If the head is inclined directly backward to an angle of 90 degrees so that the face looks directly upward, the rotation tendency will throw his body in the opposite direction,—*i.e.*, he will fall to the right (Fig. 176).

In acute suppurative labyrinthitis the nystagmus is of the same type as that induced by syringing the normal ear with cold water. The caloric test enables one, therefore, to investigate the ataxia accompanying the nystagmus seen in acute labyrinthine disease. Here, the nystagmus being rotary and falling therefore more nearly in the vertical plane, the patient, standing with head erect, tends to fall in the direction opposite to the quick eye movement (Fig. 177). In acute suppurative labyrinthitis, the nystagmus is practically always rotary and toward the sound ear, and hence the patient falls toward the diseased ear.

Pointing Tests.—Closely related in origin or causation to the reaction movements (falling directions) are the phenomena brought out by the so-called pointing tests. The normal individual, with eyes closed and having located with a finger some fixed object by the sense of touch, can move his hand upward or downward in the horizontal plane and bring his finger again into contact with the object touched, or will miss it only by a fraction of an inch. This may be called normal accuracy. In an individual, on the other hand, who exhibits the symptom-complex of vestibular irritation (*e.g.*, after rotation or the caloric test), pointing accuracy is lost, the hands in attempting without the aid of sight to move accurately in the vertical plane regularly diverging *in the direction opposite to that of the nystagmus present.* (For the theory and application of these important tests, see Chapter XIV.)

From this short preliminary discussion of vestibular phenomena we must pass to a consideration of the lesions upon which they throw light. The subject of vestibular irritation is so many-sided and presents so many phases which are of practical or academic interest to the otologist, that it is no easy matter to condense one's discussion into the narrow space limits

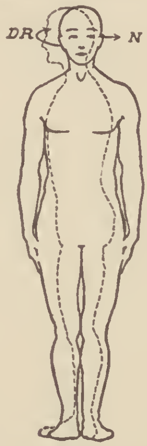

Fig. 174.

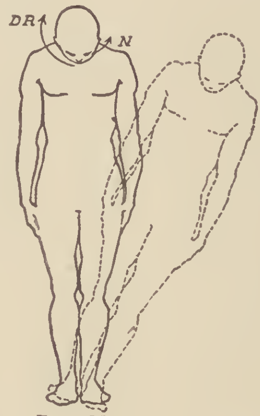

Fig. 175.

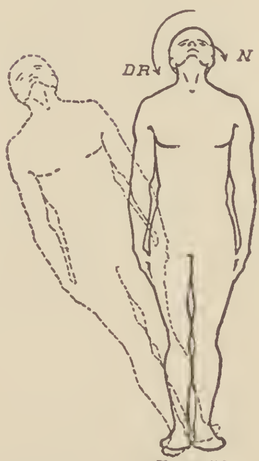

Fig. 176.

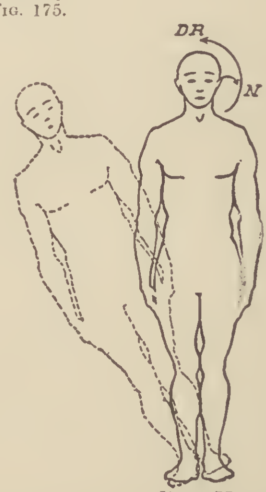

Fig. 177.

Figs. 174, 175, 176 and 177.—Diagrams showing falling directions in relation to vestibular nystagmus. *N* arrow, direction of nystagmus; *DR* arrow, direction of rotation or falling.

of a manual of this character. Naturally, this is possible only by the elimination of much which one would like to include were more ample space at his command.

The student who wishes to study for himself the variations in vertigo and ataxia in response to different forms of vestibular nystagmus, may do so by observing a person who has just been rotated upon a

revolving chair. In order to facilitate such studies, we append below a synopsis of Barany's[3] rules for determining in advance the form of nystagmus which shall follow rotation.

"Sitting erect upon a revolving chair a person revolves about a vertical axis. If we now imagine his eye cut through in a horizontal plane,—*i.e.*, by a plane at right angles to the axis about which he revolves,—it is evident that this section will describe a line upon the cornea which will vary according to the position of the head. This line will indicate the form of the nystagmus (Fig. 178). Thus, with head erect, the horizontal plane in bisecting the eye will form a line passing horizontally across the cornea, and produce a horizontal nystagmus (Fig. 178, A). With head bent laterally toward the

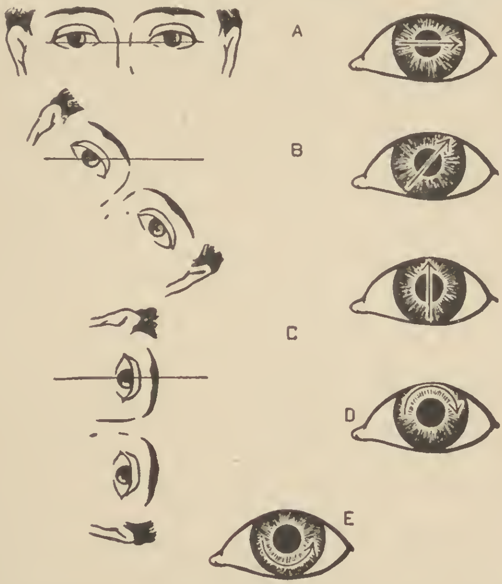

Fig. 178.—Diagrams showing different forms of rotation nystagmus.

shoulder so as to form an angle of 45° with the vertical, a horizontal section will be indicated by a line passing obliquely across the cornea, and produce an oblique nystagmus (Fig. 178, B). If the head is bent fully toward the shoulder so as to form an angle of 90° with the vertical, the eye will be bisected in a plane at right angles to its transverse diameter, and give rise to a vertical nystagmus (Fig. 178, C). With the head bent forward so that the face looks directly downward, the horizontal plane would divide the orbit so as to remove a segment which would include the iris. It should, therefore, be indicated by a circular line about the iris. The character of the nystagmus, however, is indicated by the points of contact at which the horizontal plane enters the orbit, and not by those at which it cuts its way out. With the head bent directly forward, therefore, a horizontal section is indicated by a curved line above the iris, and turning to the right will be followed by rotary nystagmus to the left (Fig. 178, D). With head bent directly backward so that the face looks upward, a horizontal section describes a curved line below the iris. With head in this position, rotation to the right is followed by rotary nystagmus to the left. But in this case the concavity of the nystagmic curve is directed upward (Fig. 178, E)."[4]

[3] Barany: Physiologie u. Pathologie des Bogengang-Apparates beim Menschen, 12-13.

[4] Kerrison: Phenomena of Vestibular Irritation, Annals of Otology, Rhinology, and Laryngology, Sept., 1909.

SUPPURATIVE LABYRINTHITIS.

Under this head are grouped such lesions of the labyrinth as are secondary to suppurative disease of the middle ear or mastoid. In the order of their importance and frequency, they may be mentioned as follows: Diffuse suppurative labyrinthitis, circumscribed suppurative labyrinthitis, diffuse serous labyrinthitis, perilabyrinthitis.

Diffuse Suppurative Labyrinthitis.—ETIOLOGY.—Suppurative labyrinthitis is always secondary to a suppurative lesion originating elsewhere in the body,—usually within the middle-ear cavity. While it may be argued upon theoretic grounds that an infective process may reach the labyrinth by other pathways,—*e.g.*, by the blood-vessels from a focus of infection within the tonsil or parotid gland, or by way of the vestibular or cochlear aqueducts from a suppurative lesion within the cranium,—it is exceedingly doubtful if such modes of infection have been definitely established in any case in which tympanic suppuration was absent.

For practical purposes, therefore, we may say that suppurative labyrinthitis is always secondary to a suppurative process within the middle ear. The spread of an infection from the tympanum to the labyrinth may occur by any one of several routes, viz.:

1. By erosion of the bone at some point upon the labyrinthine wall giving rise to a so-called labyrinthine fistula. The points at which such fistulæ are seen may be mentioned in accordance with the frequency of their occurrence in the following order,—(a) horizontal semicircular canal, (b) oval window, involving necrosis of the stapedial foot-plate or membranes, and (c) some point upon the promontory.

2. By extension of the inflammatory process through the bone, or by way of the minute anastomotic vessels without the production of a demonstrable fistula.

3. Through invasion of the labyrinth from a deep-seated extra-dural abscess on the posterior surface of the petrous pyramid (Hinsberg). This pathway of infection is considered here because an extra-dural abscess at this point is usually of otitic origin.

4. Infection of the labyrinth as a result of injury to the labyrinth wall during operation. Mygind[5] states that he has seen a considerable number of such cases, and records his belief that labyrinthine suppuration occurs more frequently as a result of traumatism incident to the radical operation than is generally recognized. That such a mode of infection must be considered seriously is emphasized by the statement of Jansen[6] that in his own operative experience suppurative labyrinthitis has occurred in 16 cases as a direct result of injuries inflicted during the radical operation, or subsequent curettage. Hinsberg[7] collected from the reports of other aural surgeons 25 cases of injury to the stapes during operation, two of which

[5] Mygind: Trans. Am. Med. Assoc., Otological Section, 1910, p. 189.
[6] Jansen: Trans. Am. Laryn., Rhin., and Otol. Society, 1908, p. 115.
[7] Hinsberg: Uber Labyrintheilerungen, p. 4.

ended fatally. To these he added a fatal case of his own. It is obviously incumbent upon us to give greater prominence to this possible source of infection than is accorded it in most text-books.

Frequency of Labyrinthine Suppuration as a Complication of Suppurative Otitis Media.—This is a question almost impossible of exact solution. Undoubtedly many deaths have occurred as a result of intracranial complications in which an intermediate lesion of the labyrinth has been unrecognized and therefore ignored. Presumably the discrepancy in the percentages of cases developing suppurative labyrinthitis reported in different clinics has been due in part to the closer study, and therefore more exact knowledge, of the disease in certain clinics as compared with others. Thus, in Prof. Urbantschitsch's clinic in Vienna, and in the University-Polyclinic presided over by Prof. Hinsberg in Breslau, where the disease has been made the subject of special study, the number of cases reported has been larger than elsewhere in Austria and Germany.

According to Hinsberg,[8] one in every 100 cases of middle-ear suppuration develops suppurative labyrinthitis. Von Stein,[9] of Moscow, in 420 cases of middle-ear suppuration operated on, found suppurative labyrinthitis in 10 cases; he therefore places the percentage as high as 2.2 per cent. Hinsberg states his belief that infection of the labyrinth is a more frequent complication of tympanic suppuration than all the intracranial complications (meningitis, brain abscess, sinus thrombosis) combined; and further that a very large percentage of all cases of meningitis and brain abscess following middle-ear disease are in reality secondary to an intermediate suppurative process involving the labyrinth. These statements are based partly upon his own observations and partly upon an analysis of the published reports of others.

Of conditions within the middle ear rendering invasion of the labyrinth probable, all writers agree that the presence of cholesteatoma is the most potent factor. Jansen some years ago reported a series of 121 cases of suppurative labyrinthitis in which cholesteatoma was the apparent cause in 71 cases. Holinger,[10] after examining the literature bearing upon this question, concludes that a majority of cases of suppurative labyrinthitis are traceable to the influence of cholesteatoma.

Tubercular lesions of the middle ear are also said to be responsible for many cases of labyrinthine disease (Siebenmann, Nager, Hinsberg). Aural tuberculosis, however, is more likely to cause widespread necrosis of the labyrinthine capsule without early signs of vestibular irritation.

While either an acute or a chronic middle-ear suppuration may lead to labyrinthine infection, by far the greater number of cases are caused by the chronic form of suppurative otitis media. On the other hand, infections of the labyrinth secondary to acute purulent otitis media seem to exhibit a greater tendency to spread rapidly to the meninges, and there-

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[8] Hinsberg: ibid., p. 1.

[9] Von Stein: Annales des maladies de l'oreille, 1896, p. 30.

[10] Holinger: Trans. Section Laryng. and Otol., Am. Med. Assoc., 1910, p. 155.

fore to end fatally. This difference is presumably due in part to the greater average virulence of the acute tympanic infections.

As to the comparative significance of different points of attack as influencing the subsequent course of the disease, experienced observers very generally agree that infection of the labyrinth through necrosis (fistula) of the external semicircular canal offers a much more favorable prognosis than do lesions in which the pathway of infection is through the oval or round window, or even through a necrotic defect in the promontory. As far back as 1907, Panse[11] emphasized his belief that fistulæ of the semicircular canals constitute a condition prognostically favorable so far as life is concerned. Somewhat in accord with this view is the statement of Hinsberg that in nearly all the fatal cases coming under his observation, one or both windows were perforated or "broken down" ("so ist in der Tat kein Zweifel darüber möglich dass bei den todlich verlaufenden Fallen fast stets eins oder beide Fenster durchbrochen sind, während Bogengangsfisteln bei ihnen sehr selter sind"). If these facts have any practical significance, they should teach us the danger of careless surgery in the neighborhood of the stapes and oval window. These are points which the student of practical otology will do well to think over and bear in mind.

Symptoms.—As the labyrinth is composed of two distinct mechanisms, —viz., the *cochlea* and the *static or vestibular apparatus*,—so the symptoms of diffuse labyrinthitis must fall under two heads,—(1) those due to impairment or loss of the cochlear function—(*i.e.*, deafness), and (2) those due to disturbance of the static or vestibular function.

Undoubtedly the most striking and characteristic phenomena of the disease are those referable to the disordered vestibule, and these will chiefly occupy our attention in the following pages. To appreciate properly these phenomena, it is necessary to recognize two distinct and clearly differentiated stages of vestibular disturbance,—viz., (a) *an acute stage*, characterized by symptoms of vestibular irritation; and (b) *a latent or quiescent stage*, characterized by vestibular paralysis.

Clinical Features of the Onset and Acute Stage.—Since suppurative labyrinthitis is practically always secondary to purulent otitis media, there may be pre-existing symptoms referable to that disease, upon which those of the labyrinthine lesion are engrafted.

The invasion of the labyrinth is usually announced by sudden and very distressing vertigo. If the patient has been previously confined to bed, as when the labyrinth is invaded during an attack of acute mastoiditis, the onset is less spectacular, and possibly somewhat less severe subjectively, than may be the case when the patient is up and about, under which circumstances he may be overwhelmed by the suddenness and severity of the attack. If the patient does not fall, he usually requires support, and, as a rule, is obliged by the severity of the symptoms to go to bed. Following quickly upon the appearance of vertigo, nausea and vomiting frequently

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[11] Panse: Arch. of Otol., Amer. edition, April, 1907, p. 87.

add to the patient's distress. Vomiting is often a persistent and frequently recurring disturbance during the first day or two of the attack.

Nystagmus.—If the eyes are observed at this time, there will invariably be seen an active rotary nystagmus with the quick eye movements toward the sound ear. The eye movements are increased in rapidity and extent when the eyes are voluntarily turned in the direction of the quick nystagmic movement, and are noticeably diminished when they are turned in the opposite direction. The nystagmus is noticeable. however, and the direction of the quick movement is unchanged, whatever the position of the eyes.

The nystagmus accompanying suppurative labyrinthitis, while usually described as rotary, is really in most cases a combination of the horizontal and the wheel types (see Fig. 166 on page 282).

Vertigo.—The vertigo also is of the rotary type, *i.e.*, the patient has the impression that surrounding objects are rotating about him, sometimes in such bewildering fashion that he is unable to analyze and correctly describe his sensations. When he is able to do so, it is found that the plane in which objects seem to rotate always corresponds to the plane of the nystagmus. When, therefore, he stands or is supported with head erect, objects seem to rotate about him in a plane approaching the vertical, but when he lies upon his back (*i.e.*, with face turned upward), the plane of the nystagmus falls more into the horizontal plane, and his sensation is of the rotation of objects in the horizontal plane about him.

The direction of seeming rotation varies; most usually it is from the side of the slow nystagmus movement. Thus, with head erect, objects seem to rise from the floor on the side corresponding to the diseased ear, and to fall or sink on the other side of his body. In some cases, however, the direction is reversed. If he closes his eyes, he has the sensation of himself rotating. If his eyes are voluntarily turned in the direction of the quick nystagmic movement, not only is the nystagmus more marked, but the severity of the vertigo and the sense of rotation are greatly increased. It has been noted (Jansen, Barany, and others) that the patient frequently assumes a characteristic position in bed,—*i.e.*, he lies with the sound ear buried in the pillow, so that, when tempted to look about the room, the movement of the eyes will be in the direction of the slow eye motion, which position tends to lessen not only the nystagmus but also the distressing subjective symptoms.

Ataxia.—If the patient stands or is supported in the upright position with head erect, he exhibits marked disturbance of equilibrium, and falls or tends to fall toward the diseased ear. Supposing, for example, that the lesion is an acute suppurative invasion of the right ear, the nystagmus will be to the left and the patient will fall to the right. If he turns his face toward the right shoulder, thus changing the plane of the nystagmus, he will fall backward. If his face is turned toward the left shoulder, he will fall forward.

The pointing reactions in the acute stage of a diffuse suppurative labyrinthitis are those invariably present during active vestibular irritation from any cause. (See Chapter XIV.)

While the patient lies quietly in bed, the subjective symptoms are minimized, and, since any sudden movement of the head tends to increase their severity, he soon learns to remain quiet and to resist any unnecessary movement.

The syndrome above described—*i.e.*, the nystagmus and associated vertigo and ataxia—is present at the onset and to some extent throughout the acute stage of practically every case of suppurative labyrinthitis. Complete absence of this triad is, therefore, a valid reason for excluding acute suppurative labyrinthitis.

Of other symptoms probably the most constant at the onset is headache in some form. Often this takes the form of severe deep-seated earache. In other cases the pain is not so localized, but is referred to other parts of the head,—*e.g.*, the vertex or occiput. The temperature is probably always elevated at the onset, and may rise to 103° or 104° F. On the other hand, the writer has seen cases of extensive labyrinthine suppuration in which the temperature at no time exceeded 101° or 102° F. When in addition to the characteristic vestibular phenomena there are frequent vomiting, high temperature, and severe headache, and these symptoms do not show early tendency to amelioration, one is forced to consider the possibility of meningeal infection. It must not be forgotten, however, that any or all of these symptoms may be present at the onset as a result of the labyrinthine lesion alone.

Cochlear Disturbance; Deafness.—A very brief statement will suffice as to the deafness of the acute stage of diffuse suppurative labyrinthitis. Usually the deafness is such as to force itself upon the physician's attention. In a case which I had an opportunity of watching from the onset to the final recovery, one of the earliest symptoms—apparently synchronous with the vestibular phenomena—was the profound deafness of the diseased ear. As the patient lay with the sound ear buried in the pillow, her failure to notice questions or even sounds originating quite near the diseased ear proclaimed its practical loss of hearing power. There was never in this case any return of cochlear function.

Personally, I have never seen an indubitable case of diffuse suppurative labyrinthitis in which very marked deafness was absent. Sudden and profound deafness is, therefore, an important sign of labyrinthine suppuration, and the retention of a demonstrably useful degree of hearing power should in my opinion be given very considerable weight as an indication that the labyrinth is not involved in a severe suppurative process.

When in a case of unilateral labyrinthine disease the deafness is in doubt, the most convenient and also the most useful and reliable test is by means of words and numbers spoken in rather loud voice close to the diseased ear, the function of the sound ear being excluded by means of the Barany noise instrument (see Fig. 79 on page 95).

Caloric Test in Relation to the Acute Stage.—In a typical case of diffuse suppurative labyrinthitis, the nystagmus toward the sound ear is due not to irritation of the diseased vestibular apparatus, but to sudden annulment of its function,—this being equivalent in effect to a direct irritation of the sound and now unopposed, vestibular mechanism. Irri-

gation of the diseased ear with hot or cold water would, therefore, be absolutely without effect upon the spontaneous nystagmus present. Irrigation of the sound ear with cold water would temporarily check the nystagmus or possibly reverse its direction for a few moments. Irrigation of the sound ear with hot water would cause great exaggeration of the nystagmus and subjective symptoms present, possibly inducing a paroxysm of nausea and vomiting. All or any of these phenomena would be more or less corroborative. The author can not see, however, that this test in the average run of cases is in any way essential to a correct diagnosis during the acute stage, and its influence upon the course of an acute inflammatory process within the labyrinth can not be regarded as free from possibilities of harm to the patient. With characteristic signs of vestibular irritation and profound deafness, the diagnosis of diffuse suppurative labyrinthitis would seem to be clear without recourse to the caloric test. On the other hand, with symptoms of vestibular irritation and retention of hearing, it would seem obviously unwise to subject the diseased ear to any shock which might possibly convert a circumscribed infection into a diffuse suppurative labyrinthitis. Unless, therefore, there are atypical or contradictory symptoms leaving the diagnosis in doubt, the caloric test is to be advised against until the symptoms of vestibular irritation have completely subsided.

Subsidence of Vestibular Symptoms.—In cases in which no intracranial complications occur, the disease runs a fairly characteristic course. The symptoms of vestibular irritation usually show rather rapid amelioration, and in their abatement follow a fairly definite order. That is to say, the vertigo and ataxia regularly subside before the nystagmus. Usually from the second to the fourth day the nausea and vomiting are relieved. From the third to the fifth day of the attack the vertigo is in many cases so much less pronounced that the patient is comparatively comfortable as he lies quietly in bed. The nystagmus, however, is still present, and turning the eyes voluntarily in the direction of the quick nystagmic movements brings a return of the vertigo. Sudden or violent movements of the head also induce recurrence. The temperature, which may be rather high at the onset,—*i.e.*, 102° or 103° F.,—usually subsides with the recession of the vestibular phenomena. At least this is the usual course in an uncomplicated case. Usually by the middle or end of the second week, and in some cases sooner, the vertigo is so far relieved that the patient—in the absence of course of contra-indicating constitutional symptoms—is able to stand without discomfort. Even now, however, sudden or exaggerated head movements are apt to induce vertigo and ataxia. Finally, during, or by the end of, the third week the spontaneous nystagmus usually completely disappears.

This describes the average, with of course very considerable variations in individual cases. The writer recalls one case of very severe and rapidly fatal labyrinthine infection in which the vertigo and nystagmus seemed completely to have disappeared within a week of the onset. In another case of extensive labyrinthine suppuration, which recovered only after

surgical removal of the labyrinth, the spontaneous nystagmus persisted until well into the fifth week.

The Use of Covered Glasses.—It has been found that vestibular nystagmus is more or less controlled, or lessened, when the gaze is fixed upon some object within easy visual range. The inhibitory influence of this focusing of the vision upon one object is not appreciable during the most active stage of the nystagmus, but becomes progressively more noticeable as the strength of the eye movements is reduced. Barany, following a suggestion of Dr. Hans Abels, makes use of this fact by placing before the patient's eyes a pair of opaque, or covered, spectacles, the surfaces of which are so near the eye that the vision can not be focused thereon. By thus removing the visual resting point, the nystagmus in many cases becomes much more marked. In this way the character of a very slight or rapidly diminishing nystagmus may be determined, or one that has apparently ceased may again become noticeable. It is well, therefore, in doubtful cases,—*i.e.*, in cases in which the presence of spontaneous nystagmus is in doubt,—to make use of this device.

When finally the nystagmus has completely disappeared, the patient no longer exhibits any demonstrable symptoms of vestibular irritation. We have now to determine whether he has suffered from (a) some form of transitory inflammation of the labyrinth which has undergone resolution, leaving an intact and functionating organ; or (b) from a suppurative labyrinthitis by which the vestibular function has been abolished. It is at this stage that the caloric test is of paramount importance.

Symptoms of Labyrinthine Fistula.—Before leaving the discussion of the acute stage, a word should be said about the so-called "fistula test." It is a recognized fact that suppurative labyrinthitis may be established with or without the demonstrable presence of a gross defect—*i.e.*, fistula—in the labyrinthine wall. It is also conceivable that defects in the bony capsule of the labyrinth may exist, as a congenital condition or even as a result of tympanic disease, without giving rise to a suppurative process within the vestibule.

In any case in which vestibular irritability is retained, the presence of a fistula leading into the labyrinth may be demonstrated by means of any instrument by which the air in the external auditory canal can be alternately compressed and rarefied. The best instrument for this purpose is one embodying the principle of the Politzer inflating apparatus, but having a smaller end piece to fit the orifice of the auditory canal. When this is moistened and pressed into the meatus, compression of the bulb condenses the air in the auditory canal and tympanum and, if a fistula is present, forces air into the labyrinth. When the static labyrinth is still functionating (*i.e.*, susceptible of irritation), this is regularly followed by nystagmus which varies in direction in accordance with the location of the fistula and the direction of the resulting endolymph displacement. The nystagmus thus induced is usually preceded by a slow movement of the eyes in one or the other direction, and this slow movement corresponds to the slow

component of the nystagmus. Compression may result in a single slow movement of the eyes and their quick return when pressure is relieved, or the preliminary slow excursion may be followed by several characteristic vestibular movements lasting some seconds. In the presence of a fistula, nystagmus can be induced either by compression or aspiration, which, however, give rise to nystagmus in opposite directions. That is to say, if compression causes nystagmus to the left, aspiration will reverse this direction, giving rise to nystagmus to the right. When no fistula exists, this experiment is either negative, or gives rise to very slight, almost imperceptible eye movements due presumably to pressure upon the structures closing the labyrinthine windows. Obviously if the vestibular irritability is completely lost, this test will give negative results even in the presence of a fistula.

The conditions upon which the compression and aspiration test may throw light may be stated as follows:

1. Chronic suppurative otitis media without labyrinthine infection and with intact bony capsule: compression test negative.

2. Defect (fistula) in bony capsule of the labyrinth, but without infection of membranous labyrinth; shown by absence of spontaneous symptoms of vestibular irritation, plus strong reactions to compression and aspiration (caloric irritability normal).

3. Acute stage of suppurative labyrinthitis with fistula; spontaneous nystagmus modified by compression and aspiration experiment.

4. Presence of fistula in the latent stage of suppurative labyrinthitis; shown by negative caloric reaction and very slight, but typical, response to compression. In this case one might infer that the vestibular mechanism had retained a vestige of functional activity which can no longer be stimulated by heat or cold, but can still react to the stronger mechanical irritation produced by compression.

Obviously when the caloric reactions are absolutely negative, the absence of response to compression or aspiration can not be regarded as disproof of the presence of a fistula, since vestibular irritability may be completely abolished.

Since it is easily conceivable that infective matter may be forced by air under pressure from an infected tympanum into an uninfected vestibule, it is clear that this test should be used with moderation and care.

Latent, or Quiescent, Stage of Suppurative Labyrinthitis.—This stage is sometimes spoken of as "chronic suppurative labyrinthitis," a term which is distinctly misleading, since it seems to imply two varieties of the disease, just as in acute and chronic purulent otitis media we have different types of tympanic inflammation. But whereas acute purulent otitis media may run its course from onset to resolution without having passed through any of the phases characteristic of chronic middle-ear suppuration, every case of diffuse suppurative labyrinthitis must inevitably progress—and usually quite rapidly—to the latent stage. The disease is said to have reached the latent stage as soon as the symptoms of vestibular

irritation—*i.e.*, spontaneous nystagmus, vertigo, etc.—have completely disappeared.

In the latent stage of the disease, the patient may be up and about, and may apparently regain his normal standard of health and strength. He may now experience no subjective symptoms referable to the labyrinth beyond the deafness and certain defects of orientation to be referred to presently. For the time being—though this is by no means invariable—the aural disease may have resumed the rôle of a circumscribed lesion having no appreciable influence over his general constitutional state. It is now of the greatest importance to apply some test by which loss or retention of vestibular irritability may be determined. The tests which have been proposed for this purpose are: (a) the caloric test, (b) the rotation test, and (c) the galvanic test.

The Caloric Test.—To those who have not made use of this test, a word as to the method of applying it may be of service. Since the reaction depends wholly upon the temperature of the water, and not in any degree upon the force employed, a slow continuous current gives better results than a stronger intermittent stream. The fountain syringe is, therefore, preferable to any form of hand syringe. Barany takes care that the water shall be at 30° C. (86° F.), having found that this temperature gives a positive reaction in the great majority of cases. There are some cases, however, in which no satisfactory reaction follows irrigation with water at this temperature. In such cases, reducing the temperature to 75° or 65° F. may induce a quick and positive reaction. The disadvantage of using very cold water is the greater likelihood of causing nausea and vomiting. We should watch carefully for the first appearance of nystagmus. To continue the irrigation after the nystagmus is clearly established adds nothing to our knowledge of the condition and will be likely to cause nausea and vomiting. Using water at about 86° F., the average time required to produce nystagmus is about 40 seconds. There are, however, great variations within the physiological limits,—*i.e.*, from 10 seconds to as much as 3 minutes. The slowest reaction in a presumably normal labyrinth which has come under the writer's personal observation was a case in which the nystagmus appeared after two minutes and fifty seconds of continuous irrigation.

As with all forms of vestibular nystagmus, the eye movements are most marked, and therefore first become noticeable, when the eyes are turned in the direction of the quick nystagmic movement. It is, therefore, well to direct the patient to keep the eyes turned in the direction of the ear not irrigated when cold water is used and to look toward the ear irrigated when hot water is employed. In a case of normal caloric reaction, the nystagmus has an average duration of 2 minutes,—differing in this respect from the nystagmus following the so-called rotation experiment, the average duration of which is about 40 seconds.

Instruments which facilitate the experiment—especially when one is working without an assistant—are Barany's "fixator" for holding the

gaze in the desired direction, and a self-retaining basin for receiving the return flow of water (see Fig. 179). An ordinary stop watch is convenient, and, if one would be exact, almost essential for determining the quickness of the reaction and its subsequent duration.

It is in the latent or quiescent stage of the disease that this test is of the greatest value, leading logically to one or other of two conclusions:

FIG. 179.—Barany's fixator and self-retaining basin.

1. If the caloric test is followed by a normal reaction, one may infer quite confidently that the labyrinth has been involved in a comparatively simple process, which, while sufficiently severe to have annulled temporarily vestibular irritability, has undergone resolution, leaving the vestibular apparatus intact. Prognosis favorable.

2. If, on the other hand, the caloric test evokes absolutely no response, one may conclude with equal certainty that the labyrinth has been involved in a suppurative process which has either (a) actually destroyed the essential structures of the membranous vestibule, or (b) has inflicted upon them such injury as to have abolished the vestibular function. While this describes a condition in which the patient may go for long periods or even indefinitely without alarming symptoms, there is no possible assurance of such immunity, and the prognosis as to the ultimate outcome can not be regarded otherwise than with anxiety.

Rotation Test.—We have seen that when a normal person seated with head erect upon a revolving chair is turned rapidly in either direction, there results when the rotations are suddenly stopped a horizontal nystagmus in the direction opposite to that in which he was turned. The average duration of this "after-nystagmus" is about 40 seconds. While this duration varies considerably within physiological limits, the duration of the nystagmus resulting from rotation in opposite directions is usually about the same,—*i.e.*, shows a difference of not more than 3 or 4 seconds. The diagnostic value of this experiment depends, therefore, entirely upon a comparison of the duration of the "after-nystagmus" in opposite directions. If, for example, an individual is turned ten times to the left and exhibits an "after-nystagmus" to right lasting 30 seconds, while ten rotations to the right result in a nystagmus to the left lasting but 15 seconds, the inference, so far as we may draw one from this experiment, is that his

left vestibular apparatus is non-irritable,—*i.e.*, that its function is destroyed or abolished.

Reducing this to a formula, we may say that *great shortening of the rotation after-nystagmus in one direction as compared with that in the opposite direction points to a non-functionating labyrinth on the side toward which the shortened nystagmus is directed.*

One can understand the rationale of this test only by reverting to Ewald's experiments. Ewald found by his experiments on pigeons that in either horizontal canal an endolymph movement toward its ampulla induced head and eye movements (nystagmus) much stronger than those caused by endolymph movement toward the small end of the canal. Barany believes that the same is true of human beings, and that the horizontal nystagmus to the right following rotation to the left is due in the proportion of two-thirds to the endolymph movement in the right canal toward its ampulla, and one-third to the endolymph displacement in the left canal toward its small end. It is clear, therefore, that if the right vestibule be destroyed, the nystagmus to the right following rotation to the left will be reduced by two-thirds, while the nystagmus to the left following rotation to the right will be reduced only by one-third. Therefore the duration of the nystagmus toward the destroyed labyrinth will be only half that toward the sound ear. Careful examination of the accompanying diagrams with the explanatory notes will, it is believed, make these points and their diagnostic significance clear.

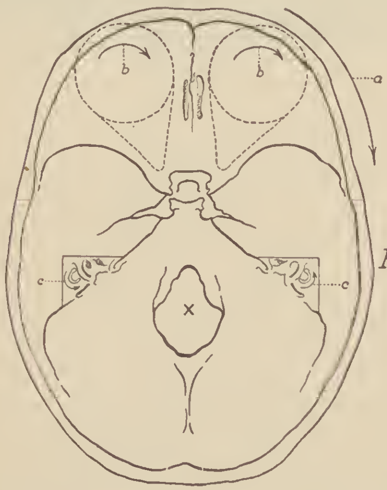

FIG. 180.—Horizontal section of skull passing through both horizontal semicircular canals. Nystagmus during rotation to right. (x) axis of rotation. Large arrow (a), direction of rotation; small arrow (c), direction of endolymph displacement caused by rotation to right; medium arrow (b), direction of nystagmus during rotation. During rotation to right there is horizontal nystagmus to right.

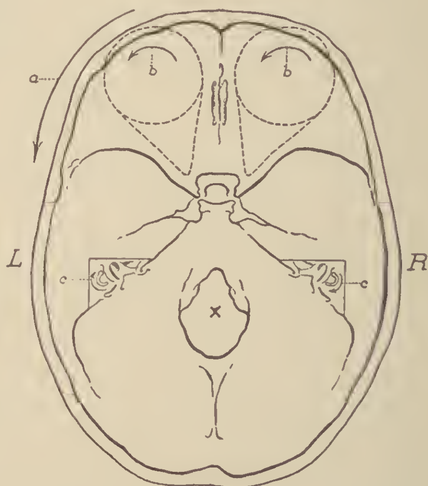

FIG. 181.—Nystagmus during rotation to left. (x) axis of rotation. Large arrow (a), direction of rotation; small arrow (c), direction of endolymph displacement caused by rotation to left; medium arrow (b), direction of nystagmus during rotation. During rotation to left there is horizontal nystagmus to left.

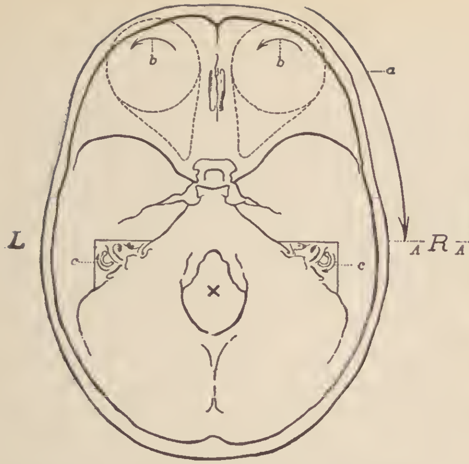

Fig. 182.—After-nystagmus (*i.e.*, immediately following rotation to right), both labyrinths being sound. (x) axis of rotation. Large arrow (a), direction of rotation suddenly checked at A; small arrow (c), reversed direction of endolymph displacement when rotations are suddenly checked; medium arrow (b), direction of reversed or after-nystagmus. Force and duration of this after-nystagmus are due to endolymph displacement in the two canals in the following proportion: by two-thirds to the endolymph displacement in the left canal toward its ampulla, and by one-third to the displacement in the right canal toward its small end.

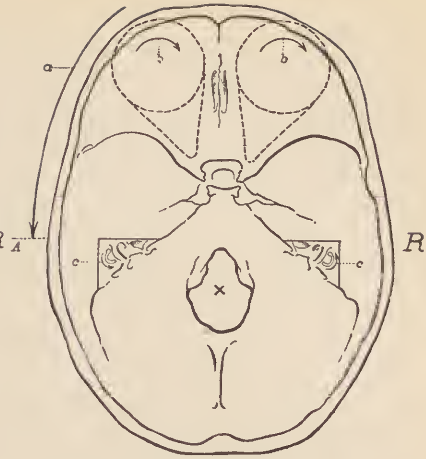

Fig. 183.—After-nystagmus (*i.e.*, immediately following rotation to left), both labyrinths being sound. (x) axis of rotation. Large arrow (a), direction of rotation suddenly checked at A; small arrow (c), reversed direction of endolymph displacement when the rotations are suddenly checked; medium arrow (b), direction of reversed or after-nystagmus. Force and duration of this after-nystagmus are due to endolymph displacement in the two canals in the following proportion: by two-thirds to the endolymph displacement in the right canal toward its ampulla, and by one-third to the displaeement in the left canal toward its small end.

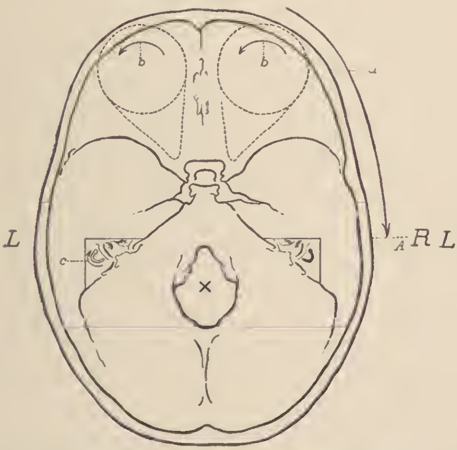

Fig. 184.—After-nystagmus (*i.e.*, immediately following rotation to right), right membranous canal having been destroyed by disease. (x) axis of rotation. Right canal (solid black) destroyed by disease. Large arrow (a), direction of rotation suddenly checked at A; small arrow (c), direction of endolymph displacement when rotations are suddenly checked; medium arrow (b), direction of after-nystagmus. Force and duration of after-nystagmus are due to endolymph displacement in left canal toward its ampulla, and represent two-thirds of the normal force; right canal, usually supplying one-third, having been eliminated by disease.

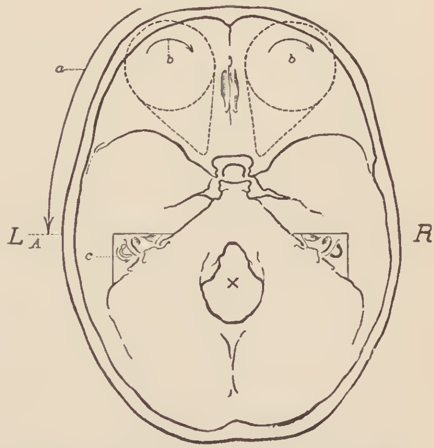

Fig. 185.—After-nystagmus (*i.e.*, immediately following rotation to left), right membranous canal having been destroyed by disease. (x) axis of rotation. Right canal (solid black) destroyed by disease. Large arrow (a), direction of rotation suddenly checked at A; small arrow (c), direction of endolymph displacement in left canal when rotations are suddenly checked; medium arrow (b), direction of after-nystagmus. Force and duration of after-nystagmus are due to endolymph displacement in left canal toward its small end, and represent therefore only one-third of the normal force; right canal, usually supplying two-thirds, having been eliminated by disease.

Therefore, the after-nystagmus toward the right is of only half the duration of that toward the left, showing loss of function in the right vestibular apparatus.

The key to the diagnostic significance of the rotation test is found in Ewald's theorem that in either horizontal canal endolymph displacement toward the ampulla causes much more forcible movements (nystagmus) than a similar displacement toward the small end. Barany accepts this fact and assumes that in man the influence of an endolymph movement toward the ampulla as compared with a displacement in the opposite direction is as 2 to 1.

The diagnostic value of the rotation test is greatest just after the symptoms of vestibular irritation have disappeared,—*i.e.* in the very early period of the latent stage. In suppurative labyrinthitis of long standing its significance and value are lessened by the fact—now rapidly gaining recognition—that as the organism gradually becomes accustomed to dependence upon one vestibular apparatus, the after-rotation nystagmus toward the destroyed or paralyzed labyrinth gradually approaches and finally equals that toward the sound labyrinth.

In the writer's opinion, the rotation test is altogether secondary in diagnostic importance to the caloric test, but it is nevertheless of some value as a corroborative test. It can be applied much more quickly than the caloric experiment, and its effects, so far as the patient's discomfort is concerned, wear off much more rapidly.

The galvanic test differs from the caloric and rotation tests in the following particulars,—viz., that, while the latter two give rise to vestibular phenomena as a result of endolymph displacement in one or other set of co-active semicircular canals, the nature of galvanic irritation is unknown, or at best conjectural. It is applied in the following way: One electrode being held in the patient's hand, the other is applied against the mastoid, or in front of the tragus, of the ear to be examined. With the cathode in contact with the ear, there results a rotary nystagmus in its own direction, —*i.e.*, toward the ear experimented upon. When the anode, or positive electrode, is held against the ear, a rotary nystagmus in the direction of the opposite ear results. These are the normal reactions, and it has been computed (MacKenzie) that a current strength of 4 ma. should induce the reaction when the labyrinth is intact and normal. Taking this as a standard of normality, the advocates of this test (MacKenzie, Alexander) claim that the requirement of a current strength of more than 4 ma. is to be regarded as an evidence of diminished vestibular irritability. This standard has not, however, been generally accepted by students of vestibular disease, and the reports of different observers as to the results of galvanic irritation both in health and disease have been so contradictory as to leave its practical diagnostic value in considerable doubt.

Vertigo of Vestibular Paralysis.[12]—I have described the latent stage of suppurative labyrinthitis as that in which all symptoms of vestibular irritation are absent. Even after the vertigo of vestibular irritation has

[12] So far as the writer knows, he was the first to describe this phase of suppurative labyrinthitis and to include it among the regular manifestations of the latent stage of the disease. Vertigo of Vestibular Paralysis: Transactions Am. Otolog. Society, 1911.

subsided, however, the patient may experience considerable inconvenience as a result of the loss of orientation sense, or knowledge of the position of his body in space, which the intact vestibular organs confer.

Orientation.—It is clear that the power of maintaining one's equilibrium in all customary positions of the body depends upon what is called orientation, or the subconscious knowledge of the position of the body in space. If through accident, or attempting the unusual, the body is thrown into positions in which its usual relations to the three planes of space are reversed, orientation becomes defective or insufficient, and the subjective disturbance known as vertigo results. Again, if the faculty of orientation is suddenly disturbed or ablated, the individual at once loses his sense of stability in space, or in other words experiences vertigo. Obviously the vertigo of vestibular paralysis is the vertigo of defective orientation.

That the vestibular organs can not be regarded as the essential organs of orientation is shown by the fact that after removal of one or both labyrinths, the individual regains in time his equilibrium. There must be, therefore, other contributory factors, and these are found in the muscular and arthrodial senses, the tactile sense, and the sense of sight.

R. T. Slinger and Sir Victor Horsley [13] published in 1906 the results of some very interesting investigations, the purpose of which was to measure the approximate accuracy of the muscular and arthrodial senses in what they called topognosis, or "the orientation of points of space." These experiments were carried out in the following manner: The person experimented upon is blindfolded, and before him is held a glass plate graduated in squares of a half centimetre each. The plate being placed in one of the three planes of space,—let us say the sagittal-vertical,—*i.e.*, a vertical plane bisecting his body anteroposteriorly,—the left hand is passively moved in different directions and the tip of its forefinger then brought in contact with some point upon the plate. He is then directed to bring the tip of the right forefinger to a corresponding point upon the opposite surface of the plate. It is obvious that in this test no information is gained through his tactile sense, which would convey the same impression wherever the finger might rest. Sight is eliminated by the blindfolding of the individual. Orientation must, therefore, be chiefly attributed to the muscular and arthrodial senses. This experiment was repeated with a large number of normal persons. It was found that there is a physiological or normal variation, or error, which varied greatly according to the position of the plate in relation to the body. Thus, with the plate held immediately in front of him in the vertical anteroposterior plane of the body and at a height about on a level with the lower end of the sternum, the average physiological variation or error was found to be 1¼ to 1½ centimetres. As the plate, still in the same vertical plane, is gradually elevated,—*e.g.*, first to a level opposite the neck, then opposite the head, and lastly above it,—the average error of orientation shows progressive and very rapid

[13] R. T. Slinger and Sir Victor Horsley: Orientation of Points of Space, Brain, April, 1906.

increase. From these variations the investigators concluded that "there is a progressive diminution of knowledge of space as we pass outward from the central axis of the body."

These experiments were repeated with 22 adolescent or young adults who had become totally and permanently blind in early childhood, and with this interesting result,—viz., that the blind subjects exhibited similar errors, which also varied in accordance with the level or height at which the plate is held; but the errors were distinctly smaller, the average error being less by one-fourth than the average error of people with normal eyes. From this they concluded, that, "if the information gained by sight is permanently blotted out, the muscular sense under necessity can by education be brought to a point at least one-fourth better than that learned by the normal person."

The above experiments have an important bearing upon the compensatory power of one part of the mechanism of orientation to assume the work of another part whose function is annulled. We have seen that in the normal man orientation is a complex function in which the vestibular apparatus, the muscular, arthrodial, and tactile senses, and sight, all take part. When sight is lost, muscular and arthrodial impressions become more sensitive and accurate. When one vestibular apparatus is destroyed, the opposite labyrinth must become accustomed to unopposed, unilateral activity, and the muscular, arthrodial, and tactile impressions of space and position must be immensely increased. Until this process of involuntary education is well advanced, it is not surprising that the individual experiences occasional vertigo and ataxia, though he does not exhibit nystagmus and may show no constant tendency to fall in any given direction.

Clinical Manifestations of Vestibular Paralysis.—We know that the onset of diffuse suppurative labyrinthitis or the removal of one labyrinth is invariably followed by very severe vertigo and disturbances of equilibrium—these symptoms being the result of hyperactivity of the sound, unopposed labyrinth. After these symptoms of vestibular irritation have completely subsided, the patient is comparatively comfortable. He has now passed the stage when sudden movements of the head cause nystagmus and its attendant phenomena, and may be up and walking about his room. On getting out of bed in the morning, however, and on making any sudden and pronounced changes in the position of his body,—*e.g.*, as in stooping over to pick up something from the floor, or even on suddenly rising from the sitting posture,—he becomes momentarily dizzy, and may even require support to prevent his falling. These symptoms are due solely to defective orientation, and, though at first occurring frequently, are very slight as compared with the constant and distressing rotary vertigo of the acute stages. The attacks rapidly grow less pronounced, and may apparently cease within a few days, so that the patient is led to believe himself cured. He is likely, however, to experience a severe recurrence on attempting any physical act to which he has not reaccustomed himself. For example, when he first attempts to walk down an inclined plane,—*e.g.*, in

going down stairs,—he may experience sudden and severe vertigo, due to the sudden call upon his still defective faculty of orientation by this new situation; and this may cause him to fall. Having had this experience, he calls into compensatory activity certain other senses,—chiefly the muscular and arthrodial senses and the sense of sight,—and soon learns to go up and down stairs without fear or danger. Again, when he first attempts to walk in the dark,—*e.g.*, in getting out of bed at night,—he may lose all sense of direction and may fall, and now must relearn to maintain his equilibrium without the aid of sight.

All the above manifestations of impaired orientation in the latent stage of suppurative labyrinthitis were demonstrated in the single case of a young physician who was under the care of Dr. A. B. Duel. The same patient, having relearned many of the common acts, such as stooping over, going up and down stairs, etc., was strolling along the street, and suddenly looked up at a tall building then in the process of erection. This deprived him of his sense of position or balance and he fell backward.

Even more typical of the vertigo of vestibular paralysis was a case which came under my care at the Manhattan Eye and Ear Hospital some years ago. The patient, a man of 28 years, had contracted syphilis seven or eight months previously, and some five weeks before I first saw him had become suddenly and absolutely deaf, first in the left and then in the right ear.

Examination revealed the following conditions: Both drum membranes practically normal; absolute deafness of both ears, both by aerial and by bone conduction. Caloric reactions absolutely negative. The patient, seated upon a revolving chair, was rotated rapidly first in one direction and then in the other, with perfectly negative results,—*i.e.*, there was no nystagmus, and the patient experienced absolutely no vertigo or discomfort. Clearly we had to deal with a patient both of whose labyrinths were absolutely non-functioning and non-irritable. Yet this patient complained as much of vertigo as of the deafness. Tests of his equilibrium demonstrated a type of ataxia presenting the following interesting contrasts with the ataxia of vestibular irritation: He walked with marked unsteadiness,—*i.e.*, with a somewhat tottering gait, keeping his eyes persistently on the ground before him. Asked why he looked on the ground he said, "to keep from falling." Yet he was able to keep a straight course and walked wherever his will directed. With eyes closed and feet approximated (Romberg), he was able to stand steadily. In walking with eyes closed, his unsteadiness was greatly exaggerated, but he still contrived to maintain a fairly straight course.

As differentiating the ataxia in this case from that characteristic of vestibular irritation, this patient exhibited no nystagmus, complained of no sense of rotation, showed no tendency to fall in any given direction. He walked laboriously and with some difficulty, compelling his muscles to carry him where his will and sight directed.

The patient was admitted as a ward patient, where his symptoms were

studied by some of my colleagues as well as myself. He never showed any signs of returning hearing, but his improvment in orientation was progressive and marked, so that when I last saw him, his gait presented no peculiarities which would attract the casual observer. In this he illustrated the rapid development of the muscular and arthrodial senses under the spur of necessity.

It sometimes happens that a case studied carefully at the time presents symptoms which are correctly interpreted only in the light of later experience. I mention by name such a patient, because her case was carefully studied by some of my hospital associates. Selena Miller, a girl then of 14 years, was admitted to the Manhattan Eye and Ear Hospital in November, 1908, with the following history. Four months previously a radical operation was performed on the right ear by a competent surgeon. The suppurative process had not been relieved thereby, and she had been readmitted to the hospital on account of failing health and atypical symptoms of disturbed equilibrium. In the hospital she rapidly lost strength and flesh, and soon became rather apathetic and practically bed-ridden. When induced to get out of bed, she could not walk without support. Support being withdrawn, she was apparently unable to maintain her equilibrium and would fall. There was no constancy in the direction of her falling, and the character of her ataxia seemed so atypical that she was suspected of being a quasi-malingerer. On careful re-examination of the ears, it was found that the right ear was absolutely deaf and gave no response to the caloric test. Operative exposure of the old wound revealed a fistula leading into the horizontal canal and granulations protruding from the open oval window. Following an operation providing free drainage from the labyrinth, she made a fairly rapid recovery with relief of all ataxic symptoms.

The interesting feature in this case was the peculiar and excessive form of ataxia, which can be satisfactorily explained only on one hypothesis: She had suffered suppurative destruction of one labyrinth; she was a delicate girl, and the effect of a rather prolonged illness had been to reduce to an unusual and remarkable degree her muscular force and control. In the latter condition, her muscular and arthrodial senses were so far below par as to have failed utterly in compensating for the disturbed labyrinthine balance. Hence the patient's inability to stand or walk, and her liability to fall in any direction according to the chance position of the body. The disappearance of the ataxia can not in this case be logically ascribed to the removal of the diseased and already non-functionating labyrinth, but rather to the relief of an extensive suppurative process, elimination of which enabled the patient to regain her muscular strength and tone, and her muscular and arthrodial senses to resume their rôle in the complex function of orientation.

The following case, which is the last I shall refer to in this connection, was in no wise unusual clinically. It will, therefore, serve the better to illustrate the point I wish to make,—viz., the great importance of recog-

nizing these defects of orientation and their resulting phenomena as belonging logically among the late clinical manifestations of the disease.

L. M., 34 years of age, a carpenter, was admitted to the Manhattan Eye and Ear Hospital in May, 1910, suffering from chronic middle-ear suppuration plus chronic suppurative labyrinthitis of the left ear. The labyrinth lesion was of long standing and the ear absolutely deaf and quite irresponsive to the caloric test. For this a combined radical and labyrinth exenteration was performed. The immediate result of the operation proved that the static labyrinth was not wholly non-functionating, for he suffered a few days of characteristic vestibular symptoms,—*i.e.*, rotary nystagmus, vertigo, etc. These quickly subsided, and the patient was soon up and about, and left the hospital to continue treatment in the dispensary. Some two months after the operation, while still under treatment for a slight discharge, the patient stated that he was in need of money and asked if he could take up his work as a carpenter, stating that he felt perfectly fit. I told him to do so, only cautioning him to make no arrangements which would prevent him coming regularly for treatment. The patient was a light-hearted, optimistic Italian, but when I next saw him he was deeply despondent. He told me that he had arranged to take up his work, but had been obliged to surrender it by the frequent attacks of dizziness which overcame him.

I had little difficulty in assuring him that his trouble was only temporary and would assuredly wear off in time. But while reassuring him, I had time to consider the rather serious possibilities of such a case. Suppose that this man had been a builder's employee whose occupation required him to perform his work while standing upon a scaffold suspended high in the air. It is not difficult to imagine what might have resulted from an attack of vertigo for which he would have been wholly unprepared. Suppose that we are called upon to operate for diffuse labyrinthine suppuration upon a boy, and, after all spontaneous symptoms have passed, allow him to resume the practice, dear to most boys, of diving into the river or swimming pool. May he not drown before he can recover from the ataxia dependent upon a sudden disturbance of orientation?

The type of vertigo which I have endeavored to describe naturally shows some variation in different individuals. Ordinarily induced only by unusual demands upon the disabled mechanism of orientation, there are undoubtedly individuals whose muscular and arthrodial senses are far below the normal average of efficiency and who after destructive labyrinthitis logically experience for a considerable period a mild grade of vertigo and ataxia of more or less constant type.

To recapitulate: The point I wish to emphasize is that there are two distinct forms of labyrinthine vertigo and associated ataxia,—one the familiar spontaneous type, due to vestibular irritation and occurring only during the acute stage of suppurative labyrinthitis; and the other not spontaneous, not constant, not necessarily accompanied by nystagmus, characteristic only of the latent, or quiescent, stage of the disease, and

induced solely by sudden and unexpected calls upon the lost or defective orientation sense, in the maintenance of which the intact vestibular organs are normally so important a factor. This type of vertigo is usually wholly relieved in time as other senses are trained to compensate completely for the lost or impaired vestibular function.

TERMINATIONS; PROGNOSIS.—Diffuse suppurative labyrinthitis may become quiescent and, having permanently destroyed the hearing and the vestibular function, may in some cases undergo gradual resolution,—possibly by obliteration of the infected labyrinthine cavities by deposition of new connective tissue. On the other hand, the suppurative process may remain quiescent during many years and then give rise to a rapidly fatal intracranial infection.

Complications.—The pathways of intracranial infection are (a) from the cochlea by way of the cochlear nerve channels into the internal auditory meatus; (b) by way of the aquæductus cochleæ to the subarachnoid space; and (c) from the vestibular cavity by the aquæductus vestibuli. By far the most frequent intracranial complication of suppurative labyrinthitis is meningitis; then come cerebellar abscess, sigmoid sinus infection, and lastly cerebral abscess. A certain percentage of cases of diffuse suppurative labyrinthitis give rise, even before the acute stage is passed, to fatal meningitis. Probably the majority of recognized cases reaching the latent stage are now operated upon. There are at present, therefore, no available statistics by which we may determine what percentage of cases of latent suppurative labyrinthitis, not subjected to operation, give rise to intracranial disease. Of this, however, there can be no doubt,—viz., that a patient who presents symptoms of chronic suppurative otitis media plus symptoms of past suppurative invasion of the labyrinth, harbors a lesion which places his life in more or less constant danger.

Circumscribed Suppurative Labyrinthitis. — Circumscribed infection of the labyrinth can be said to exist only when one or other main division of the labyrinth can be shown to have escaped serious involvement. Thus a suppurative process confined either to the cochlear or to the vestibular portion would come under this head. Theoretically, an infection of the cochlea, not reaching the vestibule, should provide an occasional example of circumscribed labyrinthitis, but it is a fact, abundantly established by clinical experience, that primary invasion of the cochlea rarely remains localized, but tends rather to rapid infection of the entire labyrinth (diffuse suppurative labyrinthitis). The static labyrinth, on the other hand, is sometimes invaded by a suppurative process which does not spread to the cochlea. Just why a suppurative lesion in this portion of the labyrinth more frequently remains localized is not easily determined. Probably these circumscribed lesions have their inception in a gradual destruction of the bony wall of one or other semicircular canal,—most frequently the horizontal,—resulting in a fistula at this point; it is possible that the gradual progress of this process is accompanied by a surrounding subacute inflammation, by which adhesions are formed, and it may be that such

adhesions act as a limiting barrier to the spread of the destructive lesion to the labyrinthine spaces beyond. However this may be, I believe that circumscribed suppurative labyrinthitis may in almost every case be defined as a suppurative lesion confined to the vestibular apparatus.

SYMPTOMS.—The most typical examples of circumscribed suppurative labyrinthitis are probably found in cases in which infection has taken place through a necrotic defect in the outer wall of the horizontal semicircular canal. The symptoms of the onset and acute stage are precisely similar to those characterizing the acute stage of diffuse suppurative labyrinthitis. That is to say, the onset is announced by spontaneous nystagmus, usually rotary and toward the sound ear, and by vertigo and ataxia, corresponding in type to the form of nystagmus present. The hearing, however, remains fairly good or is only moderately impaired. This preservation of cochlear function is in reality the differential point between the circumscribed and diffuse lesions.

The manifest symptoms of vestibular irritation undergo progressive amelioration and may completely subside within ten days to two or three weeks. The patient now presents no conspicuous vestibular symptoms, having reached the latent stage, corresponding to the latent or quiescent stage of diffuse suppurative labyrinthitis.

The acute symptoms having subsided, it becomes necessary to investigate the condition of the static labyrinth. Usually the caloric reactions are quite negative. In this case the rotation test usually shows greatly shortened after-nystagmus in the direction of the diseased ear. In certain cases, however, there is a partial recovery of vestibular irritability, the caloric tests being followed by reactions of diminished force and shortened duration. When vestibular irritability is completely absent, there is usually no return of vestibular symptoms; whereas with partial restoration of caloric irritability, the patient is not infrequently subject to very distressing attacks of recurrent vertigo. There is always a possibility in these cases that the hearing, though not profoundly affected at the time of the acute symptoms, may later undergo gradual and very pronounced impairment.

It must not be implied from the brevity of this discussion that a suppurative process confined to the vestibular apparatus is not a very serious lesion. Obviously, such a lesion may at any time spread to the cochlea. It may also give rise to meningeal infection. There can be no question, however, that so long as the cochlea escapes infection, the danger of serious intracranial involvement is distinctly less imminent than when the cochlea is also diseased. For this reason, and in order that every rational effort may be made to prevent its conversion into a diffuse suppurative labyrinthitis, it is of great importance that the circumscribed nature of the lesion should be determined as early as possible.

Diffuse Serous Labyrinthitis.—As compared with the suppurative form, serous labyrinthitis is a rare lesion. As with the former, it occurs during the course of a suppurative middle-ear lesion. With serous as with diffuse

suppurative labyrinthitis, the symptoms are dependent upon ablation or suppression of the vestibular and cochlear functions. During the acute stage the symptoms of the two lesions are practically identical, and the diagnosis of serous labyrinthitis can not therefore at this time be positively made. In the latent, or quiescent, stage, the symptoms diverge, from the fact that in suppurative labyrinthitis the loss of vestibular and cochlear function is practically always permanent, while in the serous form of the disease resolution is followed by partial or complete restoration of function.

The symptoms of the onset and acute stage are characterized by sudden and intense vertigo, with rotary nystagmus, which may at the very start be toward the diseased ear, but quickly changes its direction toward the sound ear. Nausea and vomiting are also commonly, though not invariably, present. Deep-seated earache and headache may be more or less distressing additions to the patient's discomfort.

From this short description it will be seen that the difficulties of differential diagnosis between the serous and suppurative lesions are at this stage practically insuperable. It has been claimed (MacKenzie) that severe headache, usually present in suppurative labyrinthitis, is commonly absent in serous labyrinthitis. The writer has seen, however, cases of destructive labyrinthine suppuration in which headache did not at any time appear to be an urgent symptom. On the other hand, a sufficient number of authenticated cases of serous diffuse labyrinthitis has not yet been observed and recorded to enable one with safety to exclude such common and easily induced phenomena as headache. The severe form of headache occurring during the acute stage of many cases of suppurative labyrinthitis which progress favorably—*i.e.*, without serious complications—is presumably due to hyperæmia or slight serous inflammation of the meninges, and there is no reason to believe that the same may not occur in the acute stage of a serous labyrinthitis.

With the subsidence of vestibular symptoms, the disease lapses into a latent or quiescent stage quite similar to that described in suppurative labyrinthitis. It now becomes possible for us to make a more positive diagnosis by means of the usual tests of vestibular function. If the caloric tests induce absolutely no response, and a shortened nystagmus toward the diseased ear follows rotation, and the negative reaction to these tests is repeated after two or three weeks, the diagnosis of suppurative labyrinthitis would seem to be positive. If, on the other hand, the caloric test shows returning vestibular irritability, the serous character of the inflammation may be assumed.

As before stated, diffuse serous labyrinthitis is, in the light of our present knowledge, a comparatively rare disease. The importance of distinguishing such a lesion from a suppurative labyrinthitis depends in great part upon two facts: (1) Serous labyrinthitis offers a comparatively favorable prognosis, both as to life and the aural function; and (2) operation upon the labyrinth is therefore clearly contraindicated.

Perilabyrinthitis.—This term is used to describe the condition in which the cellular bone surrounding the labyrinthine capsule is acutely inflamed. It may occur either in acute mastoiditis, or during acute exacerbations of chronic otitis media. Presumably the inflammatory process involving the cellular bone surrounding the labyrinth involves also to some extent the labyrinthine capsule itself. On this hypothesis, the vestibular symptoms may be attributed to intralabyrinthine congestion secondary to the inflammatory changes in the bone. The occasional presence with perilabyrinthitis of facial paresis or paralysis is presumably due to pressure upon the nerve by inflammatory exudates within the facial canal. The lesion may undergo resolution after the mastoid or tympanic disease is controlled, or may give rise to serous labyrinthitis or even to diffuse suppurative labyrinthitis.

Symptoms.—In typical cases, the disease is announced by sudden attacks of characteristic vestibular vertigo, which may last but a few minutes or an hour or more, but which show a marked tendency to recurrence. The first attack may be induced by some sudden movement of the head, as in stooping low, sudden rising from the recumbent position, etc., or it may occur spontaneously,—*e.g.*, during the night, awakening the patient from a sound sleep. The accompanying nystagmus is usually of rotary character and is commonly directed toward the diseased ear. Hearing during the attack may be moderately or very markedly impaired. As the attack wears off, the hearing power improves, and may reach the average acuteness for the patient. Between the attacks Weber's test is usually referred to the diseased ear (Neumann). During the intervals between the attacks the caloric reactions and the reactions to the tuning experiment are normal. With the recurrence of the attacks, their character may change in that the direction of the nystagmus may be reversed—*i.e.*, may be directed toward the sound ear—and the loss of hearing may be much more pronounced. Such changes, however, suggest the conversion of the lesion into a diffuse labyrinthitis, serous or purulent. According to Fletcher, the labyrinthine symptoms in perilabyrinthitis clear up after the tympanomastoid lesion has been corrected,—*i.e.*, after a simple mastoidectomy or a careful radical operation according to the indications present.

In reviewing the foregoing pages, it may seem to the reader that the dividing line between these lesions is in many cases so indefinite as to be of academic interest rather than of practical importance. Thus, a circumscribed labyrinthitis may easily become diffuse, a serous labyrinthitis may become purulent, and a perilabyrinthitis may give place either to serous or to purulent inflammation of the labyrinth. This narrow boundary line between the different lesions renders their clinical recognition the more important, since the indications for treatment are quite different, and the correct diagnosis and management of a comparatively simple lesion may preclude the development of one in the highest degree dangerous as to life. The more important clinical differences between the four lesions above described are given in the following table:

	Diffuse Suppurative Labyrinthitis.	Diffuse Serous Labyrinthitis.	Circumscribed Suppurative Labyrinthitis.	Perilabyrinthitis.
Onset and acute stage....	Onset sudden,—usually during acute mastoiditis or chronic suppurative otitis media,—or may result from injury to labyrinth wall during radical operation.	Onset sudden, but often preceded by short recurrent attacks of vestibular vertigo (*i.e.*, often preceded by perilabyrinthitis).	Sudden onset, secondary usually to chronic suppurative otitis media.	Gradual development, characterized by recurrent attacks of vestibular vertigo and ataxia, of short duration. Occurs during acute mastoiditis or exacerbation of chronic suppurative otitis media.
Onset and acute stage....	Absolute deafness.........	Deafness usually profound..	Hearing only moderately impaired.	Hearing during attacks impaired. Between attacks may regain normal standard.
Onset and acute stage...	Nystagmus, sometimes horizontal at onset, soon changing to combined horizontal and rotary type; directed toward sound ear.	Nystagmus rotary, or combined horizontal and rotary type, toward diseased ear.	Nystagmus, sometimes horizontal at onset, soon changing to combined horizontal and rotary type; directed toward sound ear.	Nystagmus rotary and toward diseased ear.
Onset and acute stage....	Fistula symptom very common.	Fistula symptom not common.	Fistula symptom very common.	Fistula symptom not common.
Onset and acute stage....	Caloric reactions lost........	Caloric reactions absent....	Caloric reactions absent....	Caloric and rotation reactions normal between the attacks.
Quiescent or latent stage*.	Caloric irritability permanently lost.	Caloric tests show returning vestibular irritability.	Caloric reactions usually absent.	Caloric reactions normal.
Quiescent or latent stage*.	Deafness complete and permanent.	Gradual return of hearing power.	Hearing power may be stationary, or show gradual improvement or loss. (Sudden absolute deafness would indicate conversion of lesion into diffuse suppurative labyrinthitis.)	Hearing power normal (*i.e.*, the individual's average acuteness).
Quiescent or latent stage*	Prognosis grave...........	Prognosis comparatively favorable.	Prognosis doubtful as to final outcome.	Prognosis, under proper treatment, favorable.

*After complete subsidence of symptoms of vestibular irritation.

Acute Hyperæmia of the Labyrinth. — Before leaving the subject of acute vestibular disease, a word should be said as to the condition described as labyrinthine hyperæmia. Since a direct anastomosis between the vessels of the tympanum and those of the labyrinth has not been determined, it is somewhat difficult to explain this condition as secondary to tympanic congestion. There are, however, occasional cases of severe vestibular vertigo, usually accompanied by loud tinnitus and lasting but a few minutes, which are difficult to explain upon any other hypothesis than as depending upon temporary changes in the local blood supply or pressure.

A personal experience of the writer may provide an illustration. Having suffered for twenty or more years with bilateral catarrhal otitis media, I have never experienced an acute middle-ear lesion. Some fifteen years ago, I awoke one morning with what seemed to me the severest form of vertigo. Everything in the room swam in circles about me in the horizontal plane. There was a loud roaring sound in my right ear. Whether the hearing was temporarily involved I do not know. The severity of the vertigo was such that it would have been quite impossible for me to have stood without support. This vertigo lasted probably ten or fifteen minutes,—it seemed much longer,—and then gradually wore off, leaving my ears apparently *in statu quo*. There has never been the slightest recurrence. The transitory character of the attack apparently negatives the possibility of the smallest hemorrhage or effusion into the labyrinth, and leaves sudden temporary hyperæmia as the most plausible theory as to its causation. As to the causes of the hyperæmia in such cases, we are very much in the dark, unless it be due to disturbances of the general circulation combined with local changes in the minute labyrinthine vessels.

While attempts have been made to describe in detail the symptoms of acute labyrinthine hyperæmia, I know of no recorded series of cases upon which data are based. Probably, were a sufficient number of cases brought under the care of a competent observer, it would be found that the vestibular phenomena of nystagmus, vertigo, and ataxia are invariably present and are co-ordinated according to Barany's laws, and that when the aural disorder is unilateral, the nystagmus is usually directed toward the involved ear.

PROGNOSIS AND TREATMENT OF DIFFUSE SUPPURATIVE LABYRINTHITIS.—Before taking up in detail the management of infective diseases of the labyrinth, there are one or two general facts which deserve brief consideration.

Suppurative labyrinthitis *per se* is not a fatal malady. If resulting fatally, death is caused not by the labyrinthine disease itself, but by some intracranial lesion to which it gives rise. The necessity in any case for immediate operation must be determined, therefore, not by the evidences of vestibular involvement, but by the danger of intracranial infection. If our experience, judgment, and diagnostic skill enable us to foretell from the physical character of the lesion and its clinical phenomena that meningeal infection is threatened or imminent, prompt surgical drainage of the

labyrinth is clearly the rational and conservative method of treatment. On the other hand, if we are able to deduce from the character and course of the symptoms a comparative freedom from such danger, early operative intervention is often distinctly contra-indicated.

We now know that in any series of correctly diagnosticated cases of labyrinthine suppuration a certain percentage of the patients will survive the attack without surgical intervention; that others of the same series will prove fatal through extension of the disease to the brain or meninges.

Hinsberg, from his own personal experience and from an analysis of the published records of other surgeons, believes that, of uncomplicated cases correctly diagnosticated and operated upon by competent surgeons, the mortality will not exceed 2 per cent., while of such cases not operated upon the ultimate mortality will reach 10 per cent. A consideration of such estimated percentages, and probably the observation of certain cases of rapidly fatal meningitis, have led some surgeons to the statement of rather hasty conclusions as to the necessity of early operation upon the labyrinth in all cases of suppurative labyrinthitis. Thus, Jansen[14] says, "If we appreciate the freedom from danger of opening the vestibule, . . . we should be able to set up the principle that as soon as labyrinth disease has been diagnosticated in one of its principal parts, the (labyrinth) operation is admissible in every case." The writer believes that such a dogma is not only incorrect in theory, but is likely in practice to lead to operations which are in many cases uncalled for, and in others actually dangerous to life. The author would emphasize the statement of Neumann,[15] that "not every circumscribed labyrinthine suppuration must become a diffuse one, for the intact portion of the labyrinth may be protected by firm adhesions until the diseased part of the labyrinth shall have entirely and spontaneously healed."

Clearly the management of these cases calls for careful and skilful surgery when surgical aid is indicated; but in even greater degree may the patient's life depend upon the physician's ability correctly to interpret symptoms and to deduce therefrom a correct plan of treatment.

Diffuse Suppurative Labyrinthitis. — The treatment of this disease varies with the stage and also with the type of the attack. It is best considered, therefore, under different headings.

I. *Typical Uncomplicated Case; Acute Stage.* — Symptoms: Nystagmus, vertigo, and disturbance of equilibrium; nausea and vomiting; earache, headache, moderate fever, absolute deafness of involved ear.

Treatment: The symptoms at this stage usually confine the patient to bed, where he should be kept until all signs of vestibular irritation have completely disappeared. The bowels should be evacuated by cathartic drugs or by enema. The patient should be placed upon fluid diet, this to be replaced by light diet as soon as the condition of the stomach will

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[14] Jansen: Trans. Amer. Laryn., Rhin., and Otol. Soc., 1908, p. 136.

[15] Neumann: Infectious Diseases of the Labyrinth, Laryngoscope, November, 1910, p. 1032.

permit. He should be advised and guarded against any unnecessary movements. He should not be allowed to get out of bed in order to go to the closet or for any other purpose. No attempt should be made at this time to investigate his control of static equilibrium. In the author's opinion, the caloric test should not at this time be applied except in certain obscure cases in which it may aid in the differentiation between a possible brain or labyrinthine lesion. In typical cases the caloric test adds little to our practical knowledge of the condition during the acute stage, and may influence the progress of the lesion unfavorably. The cleansing of the ear at intervals should be accomplished with the least possible disturbance of the patient. In a word, he should be subjected to no measures of examination or treatment calling for unnecessary movements of the head, which invariably add to the severity of the vestibular symptoms.

The question of operating upon the labyrinth during the acute stage of suppurative labyrinthitis should be decided solely by the presence or absence of symptoms pointing to danger of intracranial infection. If excessive and continuous headache, high fever, mounting rather than receding from day to day, frequent and persistent vomiting,—if these symptoms by their severity and particularly by their continuance point to dangerous meningeal congestion, we may be forced to drain the labyrinth in the hope of checking the spread of the disease.

On the other hand, if the clinical picture does not indicate impending intracranial infection,—*e.g.*, if the temperature is normal, or, having been elevated, shows progressive, though gradual, recession toward the normal line; if the patient is reasonably comfortable, and from day to day becoming more so; and if the symptoms of vestibular irritation are gradually subsiding, the writer believes that surgical intervention, for the time being, is distinctly contra-indicated, and should not be thought of until all symptoms of vestibular irritation have completely subsided.

II. *Diffuse Suppurative Labyrinthitis with Fistula; Acute Stage.*—Symptoms: Phenomena of vestibular irritation (nystagmus, vertigo, etc.) plus fistula symptom (nystagmus modified by compression and aspiration test); temperature normal or only slightly elevated; absolute deafness of diseased ear.

The treatment is practically the same as for Group I. The presence of a fistula of spontaneous development and presumably leading into one of the semicircular canals is generally regarded as influencing the prognosis favorably (Hinsberg, Panse, Jansen). The labyrinthine operation is, therefore, contra-indicated during the acute stage except in the presence of symptoms of threatened intracranial disease.

III. *Suppurative Labyrinthitis following Stapedial Injury.*—Characterized by symptoms of vestibular irritation and profound deafness following quickly upon accidental injury to the stapes during a radical or other tympanic operation,—*e.g.*, subluxation of stapes, rupture of annular ligament, etc.

Most observers agree that infection of the labyrinth resulting from

surgical injury in the region of the oval window is particularly prone to spread rapidly to the brain or meninges. Pus thus admitted reaches the vestibule and cochlea before any inflammatory products are formed to bar its passage along the minute channels uniting the labyrinthine and brain cavities. Such lesions also almost invariably cause absolute and permanent deafness. The author, therefore, believes that they constitute an exceptional group in which the labyrinth should be opened and drained as soon as the diagnosis of labyrinth infection is made.

As bearing upon the importance of early operation in these cases, the statistics of Jansen are of interest. His report[16] included 19 cases of accidental injuries, made during the radical operation, subsequent curettement, etc. Of these the labyrinth was subsequently opened and drained in 13 cases, of which 9 recovered. Of 5 cases in which the labyrinth was not operated upon, all died. The extreme gravity of infections of the labyrinth resulting from accidental injuries during operation is indicated by the total mortality,—viz., 9 out of 19. Jansen's report is also impressive as showing the importance of care to avoid such surgical mishaps.

Note.—It should be understood that under Group III the writer includes only those cases of traumatic labyrinthitis which result from injuries involving the stapes. The accidental opening of the horizontal semicircular canal may not give rise to diffuse suppurative labyrinthitis, in which case it belongs to a quite different class.

IV. *Suppurative Labyrinthitis with Meningeal Irritation; Acute Stage.*—Symptoms of vestibular irritation plus continued high temperature, persistent headache, frontal or occipital, and vomiting. These latter symptoms, while not very uncommon at the onset, usually show progressive diminution even during the first days of the attack in uncomplicated cases. When they persist undiminished, or are increased from day to day, one is forced to infer at least meningeal congestion of rather high grade. In such cases the labyrinth should be opened and drained and the meninges should be exposed in the posterior fossa, *i.e.*, by removing the bony plate covering the cerebellar dura from the anterior border of the sigmoid sinus forward toward the petrous bone.

It has frequently been argued that intracranial complications might be forestalled by routine early operation,—*i.e.*, early drainage of the labyrinth,—as soon as suppurative labyrinthitis has been diagnosticated. This argument is invalidated by the fact that the labyrinth operation itself may precipitate a spread of infection beyond the confines of the labyrinth,—*i.e.*, to the brain or meninges. There can be no doubt that this is far more likely to occur when the operation is performed in the early days of a labyrinthine attack than when it can be postponed until the disease has reached the latent or quiescent stage.

Latent Stage of Diffuse Suppurative Labyrinthitis.—Symptoms: Absolute deafness; absence of caloric irritability; after-rotation-nystagmus toward diseased ear only half the duration of that toward the sound ear.

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[16] Jansen: Trans. Amer. Laryn., Rhin., and Otol. Soc., 1908.

Occasional disturbance of equilibrium due to loss of orientation sense. Usually persistent aural discharge.

Treatment: In this condition we have to deal with a lesion which has already destroyed the cochlear and vestibular functions. It is a condition which imposes a very considerable responsibility upon the physician. If he could satisfy himself that the disease had run its course, setting up barriers against the further invasion of surrounding structures, it might be justifiable to treat such cases expectantly. There are, however, no means of determining that such a lesion is, or will remain, so limited. The middle-ear disease is still subject to exacerbations, and in any one of these recurrent attacks, the already exposed labyrinth may have to bear the brunt. I believe, therefore, that the safest method of treatment, and the only one which effectually removes a very positive menace to life is by the combined radical-labyrinth operation.

In many cases, aside from the dangers above referred to, the continued discharge and the dread engendered by the recent vestibular attack constitute a rather urgent reason for the adoption of some plan offering reasonable promise of a complete cure. Again the radical-labyrinth operation affords the only means to this end.

Over this question—*i.e.*, whether in a case of diffuse suppurative labyrinthitis which seemingly has run its course, leaving a functionally dead labyrinth, a radical mastoid operation, if indicated, should always be supplemented by surgical drainage of the labyrinth—there has been a growing diversity of opinion. The old dogma of the Vienna School, that in such a case any operation upon the mastoid which does not include surgical drainage of the labyrinth subjects the patient to increased danger, now finds many dissenters. Many New York surgeons of reputation now hold that there are cases in which in the presense of a functionally dead labyrinth, with sufficient time having elapsed for the labyrinthine lesion to have undergone resolution, and with no physical evidences of a labyrinthine fistula, one is justified in doing simply a careful radical operation, without opening the labyrinth. In support of this view, the fact is adduced that certain competent surgeons have opened the labyrinth in such cases without noting any macroscopic evidences of labyrinth infection; and the further fact that many cases have now been treated surgically without opening the labyrinth with apparently perfectly satisfactory results. These views and the clinical reports on which they are based are certainly worthy of careful consideration. Personally I have not up to the present time been able to accept in principle the hypothesis on which the above treatment is based for the following reasons:—

(1) To withhold operation in the labyrinth because of the absence of a visible defect in the labyrinthine capsule is possibly to overlook the most dangerous type of case,—*i.e.*, one in which a latent focus of intra-labyrinthine infection persists and in which no natural pathway of escape to the tympanum is present.

(2) To operate on the labyrinth only in those cases in which a fistula, or

necrotic defect, is disclosed during a radical operation, seems illogical from the fact that the commonest cases of demonstrable fistulæ—*i.e.*, in the horizontal semicircular canal—are precisely those which are regarded by many competent observers (Hinsberg, Panse) as offering the least danger to the patient's life.

(3) Assuming in a given case that a radical operation without surgical drainage of the labyrinth is quite satisfactory in its immediate results: is the future safety of the patient so well provided for or assured?

(4) There can be no doubt that the latent stage of a diffuse suppurative labyrinthitis is the stage in which operative intervention is attended with least risk. Is not this risk very small as compared with the possibility of a latent focus of intralabyrinthine infection which may be rekindled either by the radical operation itself or by other causes later in life?

(5) *Personal Experience.*—I have operated on a fair number of cases of diffuse suppurative labyrinthitis in the "chronic", or latent stage, without a single mishap or fatality. Conversely, in a case of chronic suppurative otitis media, with a functionally dead labyrinth, in which I performed a radical operation without opening the labyrinth, the patient redeveloped an active suppurative labyrinthitis which led to the formation of a cerebellar abscess, this causing the patient's death. This patient was 60 years old, the original invasion of the labyrinth had occurred many years previously, and the labyrinth walls showed no macroscopic defect during the radical operation.

The whole question of labyrinthine surgery is one in which dogmatic statement should be avoided, since what is safe in one man's hands is not so in another. Any labyrinthine operation by a man who is not sure of his anatomy, who has not a clear conception of the mechanical result he wishes to accomplish, and who has not been at some pains to develop his technic, is necessarily extremely dangerous. There is also a choice of operations as involving different degrees of risk. The use of the chisel in the region of the labyrinth is surely dangerous. I believe that I have evolved, not a new operation, but a simple surgical method of opening an infected labyrinth, which in the latent stage involves little or no risk to the patient's life. On the other hand, any operation in the acute stage is attended with danger which the method employed cannot eliminate. If we act on a hypothesis of supposedly greater conservatism, and operate in a case of latent suppurative labyrinthitis without draining the labyrinth, the patient may recover perfectly. But, if an active labyrinthitis is rekindled, the danger to life is many times increased, and operative intervention, if called for, offers a much less favorable prognosis.

Treatment of Circumscribed Suppurative Labyrinthitis.—*Acute stage* characterized by vestibular nystagmus, vertigo, and ataxia, earache, normal or moderately elevated temperature, nausea and vomiting at onset. Hearing only moderately impaired.

The treatment is the same as for the acute stage of diffuse suppurative labyrinthitis,—*i.e.*, absolute rest in bed, fluid or light diet, regulation of bowels, local cleansing treatment of diseased ear. Operation upon the labyrinth is positively contra-indicated except in the presence of rather pronounced and persistent symptoms of meningeal irritation.

Latent Stage.—Circumscribed suppurative labyrinthitis may in the quiescent, or latent, stage show clinical differences justifying a division of cases into two groups.

Group I, characterized by total, permanent loss of vestibular irritability (absence of caloric reactions) plus retention of a useful degree of hearing power.

The conservative treatment of such a case calls for a very careful radical operation. The region of the horizontal canal should be closely scrutinized for evidences of localized necrosis (fistula). Finding a necrotic tract leading toward the vestibule or canals, the diseased bone should be carefully removed, an effort being made to avoid injury to the membranous labyrinth beyond the area of osseous necrosis. The posterior wound should not be closed at the time of the bone operation, the plastic work upon the membranous canal being postponed until the site of the fistula has been closed or filled in by healthy granulations.

Should absolute deafness follow quickly upon the bone operation, and particularly should symptoms of meningeal irritation supervene, the safety of the patient might call for prompt surgical drainage of the labyrinthine cavities.

Should close scrutiny reveal no area of necrosis leading toward the labyrinth, the operation should be completed as a very careful "radical." While this does not cover all possible sources of danger, it contemplates a reasonable division of risk, and avoids possible harm to the patient through what is sometimes not inappropriately called "meddlesome surgery."

Group II.—Symptoms: Vestibular irritability diminished but not lost; useful hearing power retained. Periods of comparative comfort alternating with recurrent attacks of vertigo.

Presumably the lesion is of the nature of a necrotic process which has destroyed a limited portion of the membranous vestibular mechanism. The patient experiences periods of comparative freedom from vestibular symptoms, and again recurrent attacks of vestibular vertigo. The vestibular attacks probably correspond with periods in which the vestibular lesion is rekindled into fresh activity or into actual advance. Only in this way can be explained a class of cases examples of which have come under the writer's observation both in this country and in clinics and hospitals abroad. These patients are much more miserable than those in whom the vestibular mechanism has been destroyed and its function permanently annulled.

Treatment: Considering the distress and suffering which this condition imposes upon its victims, and also the dangers which are inseparable from the recurrent attacks, the writer believes that the only adequate

treatment is one which, unfortunately, sacrifices the hearing of the diseased ear,—viz., the radical operation combined with surgical drainage of the labyrinth.

Treatment of Diffuse Serous Labyrinthitis.—The treatment of serous labyrinthitis can be dealt with very briefly. During the acute stage—*i.e.*, while active symptoms of vestibular irritation are present—the disease cannot with certainty be differentiated from suppurative labyrinthitis, and the treatment is the same as for an uncomplicated case of that disease.

After the vestibular symptoms have completely subsided (quiescent stage) the simpler character of the lesion is made known by returning vestibular irritability. Obviously labyrinthine operation is not only uncalled for but contra-indicated. It is equally clear that the pre-existing tympanic or mastoid disease should be corrected, and for this reason a radical operation or simple mastoidectomy may be necessary.

Treatment of Perilabyrinthitis.—Since the symptoms of this disorder are dependent chiefly upon acute inflammatory changes in the cellular bone surrounding the bony capsule of the labyrinth, the logical treatment should be the correction of this underlying cause. In a majority of cases the attack is secondary to acute mastoiditis, less frequently to an acute exacerbation of chronic suppurative otitis media. Quite recently the writer had under his care a typical case, with nystagmus, pronounced vertigo, nausea and vomiting, in a patient suffering from acute purulent otitis media. This patient made a perfect recovery with no other treatment than incision of the drum membrane and the usual treatment for acute middle-ear suppuration. When perilabyrinthitis occurs as a complication of acute mastoiditis, the treatment involves a dual problem,—(a) relief of the mastoid lesion, and (b) avoidance of any unnecessary jar, or concussion, which might convert a simple labyrinthine congestion into an acute inflammatory process leading quickly to diffuse suppurative labyrinthitis.

If the mastoid symptoms are of moderate grade, it is much safer to depend for the time upon free incision of the drum membrane and the so-called abortive method of treatment described under acute catarrhal otitis media, leaving the question of mastoid operation to be decided later. Under this expectant plan, it will be found that many cases will make very rapid progress toward recovery. It is of course important, however, that the physician keep the patient under close observation and be prepared promptly to open the mastoid should the symptoms either of labyrinthine disturbance or of the lesion within the mastoid become more marked.

If the mastoid disease itself is of such a character as to demand prompt surgical relief, the greatest care should be observed to avoid any concussion or jar of the temporal bone. It is clearly desirable, therefore, that the mallet and chisel should not be used. The best operative method for these cases is that described by Dr. W. S. Bryant, by which the mastoid tip is first removed by means of a suitable rongeur, the same instrument

being used to remove the entire cortex. Entering the mastoid by this route, it is perfectly practicable to complete the operation with no other bone-cutting instruments than the usual mastoid curettes and rongeurs. In America this method of opening the mastoid has become the routine practice of many surgeons of note, among whom may be mentioned Drs. T. Passmore Berens and Arthur B. Duel, of New York. Bryant's description of this operation is short, clear, and contains a convincing statement of its advantages over other methods in certain cases.[17]

To epitomize: While the logical treatment of perilabyrinthitis is directed against the surrounding zone of inflammation, the time of preference for surgical intervention is the period between labyrinthine attacks. There are cases in which the attack may be aborted, or the lesion controlled, by non-operative measures. When surgical intervention during, or closely following, the labyrinthine attack is clearly indicated by the urgency of the symptoms it is important that some operative method be adopted by which surgical or bone concussion can be as far as possible eliminated.

Differential Diagnosis.—There are few conditions not directly dependent upon aural disease for which suppurative labyrinthitis is likely to be mistaken. Cerebellar abscess in certain cases gives rise to rotary nystagmus, vertigo, and ataxia, but usually there are differences in the relation of these phenomena to each other and to other symptoms present which should enable a trained and careful observer to recognize their origin. On the other hand, the physician or aurist who has only a superficial knowledge of labyrinthine phenomena might easily be led into a mistaken diagnosis, and from this to an unfortunate, and perhaps disastrous, error in treatment. It is hoped that a careful reading of the foregoing pages may be of value not only to students and practitioners of medicine, but also to special students of otology, in placing before them a sufficiently comprehensive picture of labyrinthine reactions, both in health and disease, to render such errors unlikely.

The more important clinical differences between diffuse suppurative and other inflammatory lesions of the labyrinth are given in tabular form facing page 312.

The differential diagnosis between diffuse suppurative labyrinthitis and cerebellar abscess is of special importance to the student of aural and brain surgery. It will be considered in connection with the symptoms of cerebellar abscess.

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[17] Bryant: The Technic of the Complete Mastoid Operation, Improved, Shortened, and Simplified through the Digastric Route, Transactions Am. Otol. Society, 1907.

CHAPTER XII.

SYMPTOMS OF INTRACRANIAL DISEASE SECONDARY TO AURAL SUPPURATION.

It is not to be expected that the otologist should bring to the consideration of brain lesions the broad and comprehensive knowledge of the trained neurologist. Necessarily, there may be aspects of a given case, patent enough to the latter, which escape the former. Yet it is probably true that any otologist in large hospital practice is obliged to assume responsibilities in the treatment of suppurative brain lesions quite as heavy as fall to the lot of the average neurologist. Cases are not unknown in which a competent neurologist called to such a case has been able to provide theories,—correct so far as they have gone,—but has not felt justified in advising the prompt and active intervention necessary to save the patient's life. On the other hand, there have been cases in which the surgeon, acting upon his own judgment, has performed exploratory operations on account of supposed brain lesions which have not been found on opening the skull. It is a pity, therefore, that all such cases should not come under the careful joint study of specialists in both branches of medicine. Unfortunately, the aural surgeon is frequently called upon to act in cases in which the patient's only chance lies in prompt intervention.

There are then certain suppurative lesions of the brain or meninges which will inevitably form part of the aurist's experience. Mentioned in the order of their frequency they would probably come somewhat in the following order: (1) extradural abscess, (2) infective sigmoid sinus thrombosis or phlebitis, (3) cerebral abscess, (4) leptomeningitis, (5) cerebellar abscess.

In any large series of cases of middle-ear suppuration, these brain lesions recur with sufficient frequency to impose a rather heavy responsibility upon the aurist. It may be well, therefore, to devote a few pages to a brief preliminary discussion of certain more or less common phenomena—including headache, vomiting, pulse changes, temperature changes, localized convulsions, localized paralyses, mental changes, and the different forms of aphasia—one or more of which are usually present in every case of meningeal or brain infection.

Headache.—With the exception of infective sinus thrombosis or phlebitis, headache in some form is present at some stage of every suppurative lesion within the cranial cavity. On the other hand, if we except purulent leptomeningitis, in which headache is usually both severe and constant, there is no intracranial lesion secondary to middle-ear suppuration in which it may not be absent during very considerable periods.

Macewen mentions severe headache as a characteristic and invariable symptom of the onset, or acute stage, of cerebral abscess. This, however,

is just the stage of intracranial disease which the surgeon most rarely has the opportunity to observe. After the abscess is fully formed,—*i.e.*, after it has taken the form of a circumscribed subcortical collection of pus,—the excruciating character of the pain usually subsides, giving place to dull, indefinite, and in most cases intermittent head pains. The writer has seen fatal cases of brain abscess in which for days the patient complained of little pain.

To this extent, then, headache may approach the dignity of a differential sign,—*i.e.*, in that it is quite often of dull, indefinite, or intermittent character in fully developed cerebral abscess, while in leptomeningitis it is usually constant and tends to increasing severity up to the time when the patient becomes stuporous or enters upon convalescence (rare). Complete absence of headache extending over considerable periods would justify the physician in positively excluding leptomeningitis.

In cases seen by the author, the headache of cerebellar abscess has been of distinctly severer and more persistent type than that accompanying cerebral abscess.

In cases presenting the characteristic features of brain abscess, the sudden development of very severe and persistent headache would render the prognosis more grave in pointing to probable extension of the disease to the meninges.

To whatever lesion the headache may be due, the part of the head to which the pain is referred furnishes no reliable indication of the site of the lesion; for either in cerebral or cerebellar abscess or in meningitis, the pain may be referred either to the frontal or to the occipital region, or less frequently to the vertex, or it may migrate from one to the other.

Vomiting.—Aside from that due to digestive disorders, vomiting may be induced reflexly by so many peripheral disorders that one should be in no haste to refer its origin to a supposed lesion within the skull. Nevertheless, it is an accepted fact that the act of vomiting is under the control of a "centre," possibly in the medulla, and excited through the medium of the vagus; and it is a reasonable hypothesis that this centre may be irritated, either directly or through association fibres, by various intracranial lesions.

Starr says that vomiting may be induced by apoplexy (initial stage), by brain tumor or abscess, or by meningitis. Gowers states that "organic disease in any part of the brain may cause vomiting." According to Macewen, "vomiting occurs in the initial stage of brain abscess," but rarely after the abscess is fully formed. Vomiting, then, may be caused by any lesion so situated as to irritate or press upon certain nerve centres at the base of the brain.

In its most typical form, central vomiting presents the following characteristics: It is projectile, is not accompanied by nausea or the usual signs of disturbed digestion, and is not influenced by remedies usually corrective of gastric disorder. While this describes the type, it is not safe to assume that vomiting is not of central origin simply because symptoms of digestive disturbance are present. The central irritation may be suffi-

ciently pronounced to induce independent contraction of the stomach with projection of its contents, or may fall short of this result, leaving the nervous control mechanism of the stomach in a state of irritable unbalance easily acted upon by slight peripheral causes.

Vomiting, with or without nausea, occurs so much more frequently with meningitis than with other intracranial lesions that, taken alone, it must be regarded as pointing rather strongly to meningeal disease.

Next to meningitis there can be no doubt that vomiting is more frequently present with cerebellar abscess than with any other brain lesion. While it rarely occurs after the onset, or formative stage, of cerebral abscess (Macewen), the author has observed several cases of cerebellar abscess in which it was a rather prominent symptom not only at the onset but recurred at intervals throughout the entire course of the disease.

Aside from its frequent association with meningitis, vomiting is a general rather than a focal symptom. In association with other symptoms, however, it may be of considerable value in determining the site or character of an intracranial lesion. Among such combinations may be mentioned the following:

1. Recurrent vomiting with nausea, accompanied by rotary nystagmus, vertigo, and ataxia, showing rapid and progressive diminution in severity, suggests suppurative labyrinthitis.

2. Recurrent vomiting without nausea, accompanied by rotary nystagmus, vertigo, and ataxia, persistent or tending to increased severity, strongly suggests cerebellar abscess.

3. Recurrent vomiting with incoördination ataxia of one or both hands, high temperature and tendency to delirium being persistently absent, suggests cerebellar abscess.

4. Recurrent vomiting with headache, fever, and tendency to delirium points almost with certainty to meningitis.

Gowers's Syndrome.—Gowers believes that persistent headache coupled with persistent vomiting, for which no discernible cause is present, and which is not relieved by any of the usual remedies, constitutes an important syndrome which in itself is very strongly diagnostic of intracranial disease.

Temperature Changes.—Moderate temperature variation may be excited by causes too various for it to be of great diagnostic value except in association with other symptoms. There are, however, certain facts which may well be borne in mind in examining the temperature chart in a case of suspected brain disease. It is probable that most suppurative brain lesions are ushered in by a rise of temperature. If, however, it be situated well beneath the cortex in the cerebral substance,—*e.g.*, a cerebral abscess,—the temperature after the lesion is fully developed may return to normal and remain so for considerable periods. In deep-seated cerebral lesions, therefore, the temperature may furnish absolutely no evidence of the actual condition. On the other hand, any form of inflammation involving the meninges or the cerebral cortex is commonly announced by an elevation of temperature which, though varying in degree, is usually more

or less continuous. A continuously normal temperature is usually sufficient, therefore, to exclude meningeal involvement; and, per contra, in suspected intracranial disease constant elevation of temperature is rather strongly suggestive of a cortical lesion,—*i.e.*, meningitis or peripheral encephalitis.

The only intracranial disease to which, after exclusion of other causes, the temperature may furnish the positive clue is infective sinus thrombosis. Naturally an infected clot, situated within a large venous channel, and from which infected particles may be periodically discharged directly into the general circulation, might be expected to give rise to a very characteristic type of temperature variation,—the intermittent type characteristic of periodic septic absorption.

Changes in Pulse-rate.—Within certain limits the pulse-rate varies in acute intracranial disease in accordance with the same laws which regulate its changes in acute disease originating in other parts of the body. That is to say, it is probable that the temperature is elevated and the pulse quickened at the onset of every acute suppurative lesion involving either brain or meninges. It is also an established clinical fact that in nearly every fatal case of acute intracranial disease the pulse is greatly accelerated shortly before the end. Between the initial and terminal stages, however, the pulse-rate may show changes of the greatest diagnostic importance.

It has been noted in many cases of brain abscess that the pulse-rate at some stage of the disease has been markedly reduced in frequency. This occurs quite as often with abscesses of the cerebrum as with those of the cerebellum. The same lowering of the pulse-rate has been frequently observed also in connection with rapidly growing brain tumors. It has also been noted in certain cases of purulent leptomeningitis. The phenomenon seems, therefore, to be due largely, but not always solely, to increased intracranial pressure.

The relation between reduced pulse-rate and such lesions has frequently been demonstrated by post-mortem findings and by surgical intervention. Furthermore, in many cases of brain abscess, the slow pulse-rate has been immediately corrected by surgical evacuation of the pus (Macewen). When, therefore, a patient suffering from suppurative middle-ear or mastoid disease suddenly exhibits a markedly reduced pulse-rate,—*e.g.*, 60, 50, or 40 beats per minute,—this symptom alone is rightly regarded as rather strongly suggestive of brain abscess.

Somewhat puzzling are certain cases of meningitis in which a reduced pulse-rate has been observed. While increased intracranial pressure is, of course, common in some forms of meningitis, it seems probable that there may be other causative factors at work in the bradycardia of such cases,—*e.g.*, an accompanying encephalitis causing perhaps irritation or excitation of some centre exerting an inhibitory influence over the heart-beats. This is apparently the view held by Gowers. Such cases, however, are somewhat exceptional. As a rule, all forms of meningitis are accompanied by an acceleration of the pulse-rate.

The slow pulse resulting from the formation of a brain abscess is usually a changing symptom, having some relation to the increasing pressure. The reduction of the pulse-rate is not, however, in direct proportion to the size of the abscess,—a very small abscess sometimes causing very appreciable slowing of the pulse. If the abscess becomes encapsulated,—*i.e.*, enters upon a latent stage of indefinite duration,—it is probable that the intracranial pressure undergoes gradual readjustment with restoration of the normal pulse frequency. On the other hand, rupture of a brain abscess with escape of pus either into the ventricles or subarachnoid space, is invariably announced by a train of alarming symptoms, among which an exceedingly rapid pulse is always prominent.

Macewen's Syndrome.—Macewen has called attention to the combination of two symptoms,—viz., high temperature associated with subnormal pulse-rate,—as strongly diagnostic of intracranial disease. The author believes that this association—even when the fever is not high and the pulse retardation is of moderate degree—is most important. It is so difficult to think of any lesion outside of the skull cavity which could give rise to this phenomenon, that he is inclined to regard it as pathognomonic, not of any particular lesion, but of intracranial disease.

UNILATERAL MUSCULAR SPASM AS A SYMPTOM OF CEREBRAL DISEASE. —General convulsions—*i.e.*, of both sides of the body—rarely occur as the result of a cerebral lesion of otitic origin. On the other hand, unilateral spasms confined to certain groups of muscles may point quite clearly to irritation by such a lesion of the motor centres of the muscles involved. The condition may consist merely of twitching of a single muscle or muscle group, or may be so severe as to justify the term localized convulsion, which condition is known as *Jacksonian epilepsy*, after the man who first described it.[1] In a typical case the symptoms occur somewhat in the following order: First a sensation of tingling in the limb or region involved, then there is an involuntary contraction of a single muscle or muscle group, this being followed by clonic spasms or convulsions, other related muscle groups being successively involved. Thus, the contractions may begin at the shoulder and gradually extend down the arm so as to include the wrist and fingers, or they may commence in the fingers and extend in the opposite direction toward the shoulder. In either case the spasms of these muscles would point to irritation of the middle third of the precentral or ascending frontal gyrus. Or the convulsion may in a similar way involve the muscles of the lower limb, which would point to irritation of the paracentral or upper third of

[1] Hughlings Jackson, of London, published in 1861 the first recorded series of cases in which the patients, having suffered from a peculiar form of unilateral spasm confined to certain muscle groups ("Jacksonian epilepsy"), were subjected post mortem to careful examination in the dead-house. The post-mortems showed uniformly lesions involving certain cortical areas of the opposite cerebral hemisphere, which he therefore assumed to be the motor centres of the muscles in which contractions had occurred. These records provided the starting-point and foundation upon which our knowledge of cerebral motor localization has been built up.

the precentral convolution. Another typical Jacksonian attack is characterized by convulsive lateral movements of the head and eyes to one or the other side,—*i.e.*, there are coincident lateral nystagmus and successive jerky movements of the head in the horizontal plane in one direction until the face looks over one or the other shoulder. This phenomenon is caused by irritation of the motor centre for the head and eyes, which is located in the posterior end of the second or middle frontal convolution. The movements are toward the shoulder opposite to the side of the lesion. In these attacks there is no loss of consciousness. The convulsion may last several minutes, and, in accordance with its severity and duration, leaves the muscles in a condition of flaccid weakness or actual paralysis, which, however, is only temporary. If such an attack has been carefully observed, much may be learned from the order in which it occurs. Thus, the premonitory tingling, which may precede the convulsion by several minutes, is usually referred to the region of the initial contraction; and the initial contraction will throw light on the initial centre of irritation in the cerebral cortex. The subsequent muscular paralysis, or weakness, will be most marked in the muscle group in which the spasms first occurred, and these muscles will be the last to recover their tone and power.

It is generally conceded that a cerebral lesion can cause muscular spasms only when so placed as to be capable of causing, either directly or indirectly, irritation of the cortical motor area. For this reason many cases of intracranial disease end fatally, or are relieved by surgical intervention, without ever having produced motor disturbances of any kind. Nevertheless, it is known that a cortical lesion primarily outside of the Rolandic area may by cortical extension involve the motor area, and it is clear that a subcortical lesion may by peripheral extension also involve the cortex. It seems evident that irritation from a subcortical lesion, acting by pressure or through its proximity to the cortex, would give rise to but slight muscular contractions as compared with an irritative lesion of the cortex itself. Nevertheless, such slighter phenomena might throw much light upon an otherwise obscure lesion. The symptoms should therefore be borne in mind in every case of suspected intracranial disease of otitic origin. When localized muscular spasms do occur in the course of a brain lesion, they point clearly to the following facts,—viz.: (a) that the lesion, if subcortical, is sufficiently peripheral to cause cortical irritation; (b) that, if cortical, it must be so situated as to involve either directly or by lateral extension the precentral, or motor, area; and (c) if the sequences of the attack have been carefully observed, they may supply further data as to its exact localization.

Unilateral Muscular Paralysis or Paresis.—The results of cortical lesions in the motor area of the brain are so generally understood as hardly to require more than passing mention here. Destructive lesions in any part of the motor area of either cerebral hemisphere are followed regularly by volitional paralysis, partial or complete, of the corresponding muscle-groups of the opposite side of the body. Thus, a paralysis of cere-

bral origin confined to the wrist or elbow muscles of either side would suggest a cortical lesion of the middle third of the ascending frontal convolution of the opposite cerebral hemisphere; while paralysis of the foot or leg muscles would point to a lesion in the upper third of the same convolution. But paralysis of the same muscle-groups may also result from any subcortical lesion within the motor tract, which includes the projectional fibres of the appropriate cortical centres. A brief reference, therefore, to the common paralytic effects of such subcortical lesions, as compared with those usually resulting from a cortical lesion of equal size, may be permissible here.

From the under surface of the motor (precentral) area, the projectional fibres converge to meet in the internal capsule and there form the densely arranged motor tract which from this point is continued downward through the crus cerebri, pons varolii, and medulla to the opposite side of the cord. It is obvious that, in their passage from the cortex to the internal capsule, the fibres from the lower third of the precentral, or ascending frontal, convolution pass more or less horizontally inward, while the fibres of the middle third must assume more oblique directions gradually approaching the vertical plane. Finally the motor fibres of the upper third pass downward and somewhat outward around the lateral ventricle to join those of the lower and middle thirds in the internal capsule. This arrangement is shown in the diagrammatic figure (Fig. 186), which also shows very graphically the varying results of lesions in the cortex and in the motor tract. The darkened areas represent destructive lesions in the cortex, internal capsule, and crus cerebri. Numbers 1, 2, and 3 represent cortical lesions in the motor areas of the leg, arm, and face respectively. It is obvious that any one of these, occurring singly, could give rise to paralysis of but one set of muscles,—*i.e.*, those of the leg, arm, or face; and that similar monoplegias might result from subcortical lesions in the centrum ovale involving the projectional fibres from single motor centres (4 and 5). If, however, we follow these projectional fibres from the various motor centres into the internal capsule, it becomes clear that a comparatively small lesion in this situation (6) would be likely to result in more widespread paralysis (hemiplegia).

While it is perhaps theoretically possible for a small lesion situated in the internal capsule to involve the projectional fibres of one motor centre only, with consequent monoplegia, this is exceedingly unlikely to occur. A comparison of the autopsy finding with the clinical records in fatal cases of paralysis due to cerebral lesions would seem to show that in the great majority of cases, lesions of the internal capsule, if sufficiently severe to intercept motor impulses, give rise to rather widespread paralysis; and conversely, that a majority of all cases of hemiplegia resulting from suppurative brain lesions are due to lesions involving the internal capsule. On the other hand, only the most extensive cortical lesion could give rise to widespread paralysis. While an abscess confined to the temporosphenoidal lobe—the usual seat of an otitic abscess—might through transmitted

pressure give rise to extensive paresis, complete hemiplegia—*i.e.*, involving equally upper and lower limbs—is rarely or never so produced. As in the internal capsule, so in the crus cerebri, pons and medulla oblongata, lesions of the motor tract give rise to paralysis coextensive with the cortical area whose motor fibres are interrupted.

From the fact that the motor oculi nerve arises from the inner side of the crus cerebri and supplies the ocular muscles on the same side as the crus from which it springs, a lesion in this situation (7) is apt to produce a

FIG. 186.—Diagrammatic picture of a transverse section of the brain. (Modified from Starr.)

characteristic form of paralysis,—*i.e.*, to involve the motor tract supplying the muscles of the opposite side of the body (hemiplegia), and the third nerve (ocular paralysis) on the same side as the lesion.

So far as we may formulate deductions from the above, they may be stated as follows:

(a) In suppurative intracranial disease, however originating, paralysis of a single group of muscles suggests a cortical lesion, or a lesion so placed in the centrum ovale as to intercept only the projectional fibres of the motor centre involved.

(b) Hemiplegia, or widespread unilateral paralysis, points with greater probability to a lesion involving the internal capsule.

(c) Unilateral ptosis with external squint and inability to rotate the

eyeball upward, inward, and downward, accompanied by paralysis of the leg, arm, and face muscles of the opposite side, points to a lesion of the crus cerebri on the side corresponding to the ocular symptoms.

MENTAL DISTURBANCES.—The relation of mental disturbances to intracranial disease is hardly within the scope of a manual of otology. The author wishes merely to refer briefly to certain forms of mental disorder as having a practical bearing upon the diagnosis and prognosis of brain lesions of otitic origin. So far as he has been able to observe, the mental symptoms occasionally characterizing these lesions conform almost invariably to one or other of three main types,—viz.: (1) *Depressed or retarded cerebration*, due usually to a lesion producing great increase in intracranial pressure; (2) *loss of cerebral control*, leading first to mental excitement and finally to delirium, due usually to an irritative lesion of the cortex or the meninges; and (3) *apraxia*, or inability to think coherently, this being due to a loss of the concepts of familiar things.

1. *Depressed or Retarded Cerebration.*—In this condition cerebration may be obscured almost to the point of cessation. It is a condition of extreme mental lethargy. As a consequence, there is more or less difficulty in commanding the patient's attention even to simple questions. Having gained his attention, he answers, if at all, correctly. Sometimes there is distinct evidence of retardation of the mental process. Having asked him a question and waited for a reply, the answer comes after a long delay, just when you have concluded that he has either not heard or not understood. The condition is frequently accompanied by a tendency to lethargic somnolence. He may ask for something, and doze off, forgetting his request, before it can be brought. Evidently, the mental processes are obscured, retarded, minimized, but neither ablated nor perverted.

This form of mental disturbance is usually the result of great increase in intracranial pressure, as in the case of a large and rapidly formed brain abscess. It is not of itself an evil prognostic sign, since it usually characterizes a lesion which may respond favorably to surgical treatment,—*e.g.*, the successful evacuation of a brain abscess.

2. *Delirium* may be of low, muttering form or of the violent type, in which the patient screams, uses wild or abusive language, and can with difficulty be kept in bed. In either case the condition is equivalent to mental oblivion, since the patient, if he regains his mental balance, never has any knowledge or memory of what has occurred.

Delirium resulting from intracranial infection usually points to a cortical lesion,—*i.e.*, peripheral encephalitis or meningitis. It is of unfavorable prognostic significance, in that it points to a type of lesion which ends oftener in death than in recovery.

3. *Apraxia* describes a condition in which the patient, though conscious and perhaps striving to think coherently, is unable to do so because he can not command clear and complete mental pictures of familiar objects. It is demonstrated when a person by the incoherence of his speech shows that he is unable to recognize or appreciate the nature or uses of things

about him. Etiologically it bears a strong analogy to the various forms of aphasia (next to be described), with some of which it coexists. In right-handed individuals it indicates a lesion in the left cerebral hemisphere and *vice versa.* It is not necessarily an unfavorable prognostic sign, provided the site and character of the lesion can be determined.

APHASIA.

The subject of aphasia is so large, and its causes so deeply rooted in neurology, that we can attempt here only a brief description of such speech defects as may occasionally occur in brain lesions of otitic origin.

By aphasia we mean loss of the power of expressing one's thoughts intelligibly in spoken words. This definition is elastic, as it should be, for the aphasic individual may be practically or nearly speechless (motor aphasia); or his words may come in disordered and meaningless sequence (paraphasia); or he may be unable to recall a familiar object from the sound of its name, and, since all name-sounds have ceased to have any mental associations, he is also unable to command the name of à familiar object seen (word-deafness). Any of these defects may result from a cerebral abscess of otitic origin.

Inability to convey one's thoughts correctly in spoken words is not infrequently associated with inability to recognize written words or symbols (word-blindness), in which case the individual would naturally be unable to write at will,—or, rather, to express his thoughts in writing (agraphia).

Again, either aphasia or agraphia may be of two main types,—*i.e.*, either sensory or motor.

The cortical centres, the integrity of which is essential to intelligible speech, are in the left side of the brain in right-handed persons, and *vice versa.*

Before attempting to trace these commoner forms of aphasia to their appropriate lesions, it may be well to recall briefly the physiological basis of coördinate speech. It will be remembered that, as with the convolutions of the brain, so the smallest cortical "centres" are brought into relation with each other and with other parts of the cerebrospinal system by three sets of fibres,—viz., (a) association fibres which pass from one centre to another, and by virtue of which the smallest cortical area is said in some degree to be associated with every other; (b) commissural fibres which pass, as in the corpus callosum and anterior commissure, from one hemisphere to the other, and by virtue of which the two hemispheres act as one organ; and (c) the projectional fibres which pass from each cortical centre to the base of the brain and spinal cord.

The time intervening between a baby's birth and its first efforts at intelligible speech is consumed not so much in acquiring the physical power of articulation, as in storing the mind with certain impressions, or "memory pictures," which are essential to coördinated thought,—this being prerequisite for coördinated speech. The lowest type of idiot can not acquire language because coördinated thought is to him impossible.

"The basis of language," Starr tells us, "is a series of memory pictures," and these memory pictures are stored in various special centres for the

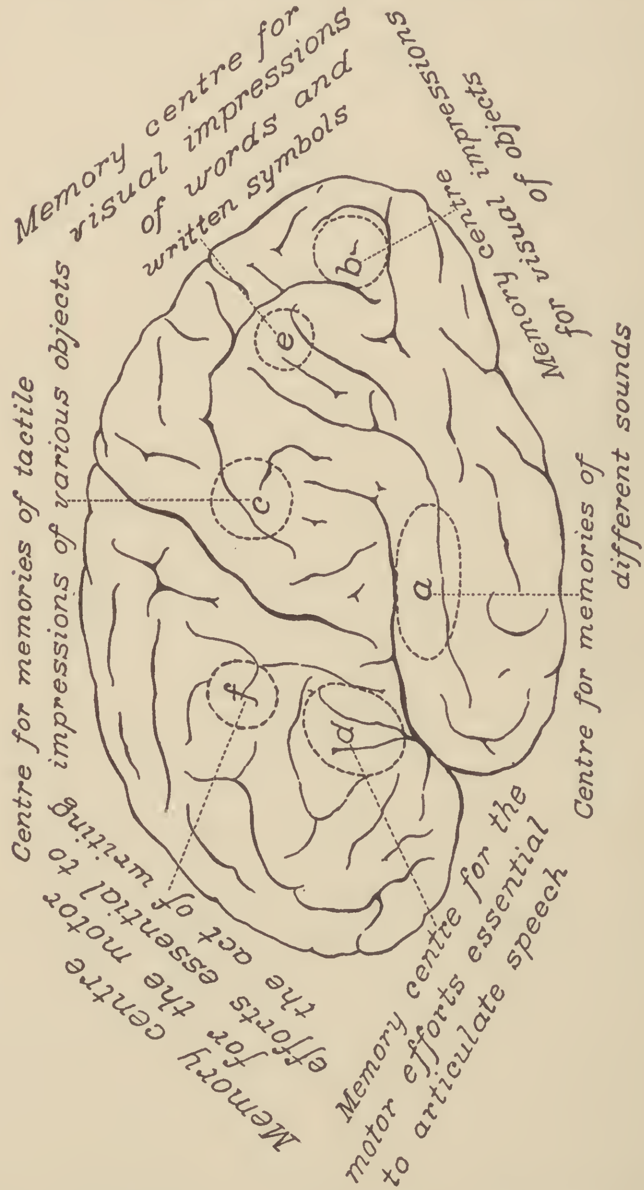

FIG. 187.—Chart showing important aphasia centres.

reception and retention of such impressions. Thus, before the child can make intelligent use of the simplest noun, he or she must have a series of memory pictures relating to it stored in various memory centres in differ-

ent parts of the cerebral cortex. Taking, for example, the word "doll," the child must have, first, a definite memory picture of the sound of this word, and this is stored in a special cortical centre for the memory of various sounds. This centre is located in the middle of the first and second temporal convolutions (Fig. 187, *a*). Next, she must have also a definite memory picture of the shape, general color scheme, and common sizes of dolls,—*i.e.*, a visual impression based upon the dolls she has seen. The special memory centre for visual impressions of various objects is located in the second occipital convolution (Fig. 187, *b*). And, third, she must have a definite impression of the texture or solidity of the various parts of the doll, this memory picture being stored in a special area for the retention of tactile impressions of various objects. This centre probably includes adjacent portions of the posterior central and superior and inferior parietal convolutions (Fig. 187, *c*). To these memory pictures—*i.e.*, of the sound of the name, the visual impression of the image, and the tactile impression of its texture and consistency—must be added a memory picture of the motor effort required to produce the articulate sound, this being stored in a special centre located in Broca's convolution (Fig. 187, *d*). It is now quite intelligible that these four memory impressions, brought into relation and coördinated through the association fibres passing between the various memory centres involved, should produce a concept, or complex mental image, and that this concept is essential to the intelligent use of the word. It is also a logical deduction that destruction by injury or disease of any of these memory centres, or of the association fibres through which their impressions are combined and coördinated, might easily produce defects of speech,—*i.e.*, some form of aphasia.

It is clear that in addition to the four "memory pictures" above described, and which probably constitute the essentials of the earliest and simplest conception of the child's mind, there are also innumerable other memory impressions relating to the object named,—*i.e.*, its uses, advantages and disadvantages of certain kinds, its associations by analogy or contrast with similar or dissimilar objects, etc.,—which come with increasing experience and advancing mental development. With the educated adult, for example, the concept of each object must include at least two additional centres for the special memory pictures which enable him to read and to write, respectively:—*i.e.*, he must have memory pictures of the appearance of written words and letters, the special centre for such impressions being located in the angular gyrus (Fig. 187, *e*); and he must have a memory picture of the motor acts required to form the letters and particular words. Destruction of the first of these—*i.e.*, the visual word centre—would render the individual unable to read. Destruction of the centre for the motor or effort memories of writing would render him unable to write, though he might still be able to read with understanding.

Obviously, with normal individuals the special senses—particularly of hearing, sight, and touch—are essential factors in the development of every complete concept. The cortical centres for the special senses of sight,

hearing, touch, etc., therefore play an essential part in the normal automatic development of speech. The congenitally blind are slow in learning to talk. The child absolutely deaf from birth never acquires language except through artificial methods of instruction.

With the above facts in mind, the various speech defects become more intelligible to us; for it is clear that we can interfere with the processes of coördinated thought and speech either by destruction or injury of one or more of the special memory centres involved, or by destroying the association fibres by which they are connected. We are now better equipped to study the different forms of aphasia as aids to diagnosis.

Suppose, for example, that we are examining a right-handed individual suspected of having an abscess in the left cerebral hemisphere. We begin by asking him very simple questions requiring chiefly affirmation or negation,—*e.g.*, if he feels any pain; if he is married; if he has children, etc., etc., receiving intelligent replies which indicate that up to a certain point his cerebration is normal.

Sensory Aphasia.—*Partial Word-deafness.*—We now show him some familiar object, as, for example, a penknife, and ask him to name it. He can not do so, and may call it a "key" or a "pencil." Ask him what it is used for, and he answers correctly,—*e.g.*, "to cut with." Ask him if it is a key, a pencil, or a penknife, and he corrects his former statement and says that it is a penknife. Now ask him the uses of articles not exposed to his view,—*e.g.*, "what is a pencil used for?" "What is a watch used for?" etc. He may be unable to answer correctly. This condition is one of sensory aphasia due to *partial word-deafness*,—partial because, though unable to name a familiar object shown him, he can, when several names are spoken for him, select the correct one. Though he may be unable to recall the uses of an object from the sound of its name alone, he can, if both the name be sounded and the object shown him, recall their relation. In other words, both the auditory and the visual memory pictures are intact, but there is a break in their association through a lesion involving the association fibres by which the auditory memory centre is brought into relation with others. In such a case, the lesion is subcortical and one which has to a great extent cut off the special centre for sound memories from its association with the other memory centres essential to intelligent speech. One would expect in such a case a subcortical lesion in the neighborhood of the first and second temporal convolutions. This, in the writer's experience, is the commonest form of aphasia in cases of brain abscess of otitic origin.

Total Word-deafness.—If the patient is unable to call the name of a familiar object shown him, though recognizing its character and uses, and, when its name with several others is spoken for him, is still unable to call the correct one, the condition is one of *total word-deafness* and presumably due to a cortical lesion involving the special centre for sound memories. Such a lesion usually produces great confusion of speech, and also renders his mother tongue almost incomprehensible to him. It is exceedingly rare as a result of suppurative brain lesions.

Intercortical Motor Aphasia.—This condition is produced by a subcortical lesion which injures, or presses directly upon, the association fibres connecting Broca's convolutions (motor speech memories) with the other memory centres. The other memory centres essential to coördinated thought being intact, and their interassociation not disturbed, the patient's cerebration is not necessarily disturbed. He understands what is said to him, and can express his thoughts in writing. He therefore makes conscious and strenuous efforts to express himself coherently in speech, but without success, the words and even syllables being transposed, and following each other without intelligible sequence. According to Starr, this variety of speech defect is produced by lesions involving the association tract between Broca's centre and the temporal auditory memory area, which tract passes beneath the island of Reil.

Word-blindness (causing Sensory Agraphia).—The special centre in which are stored visual memory pictures of objects seen and visual centre for written symbols,—*i.e.*, written or printed letters, words, etc.,—are not identical,—the former being located in the second occipital convolution, and the latter in the angular gyrus (Fig. 187, *e*).

It is possible that an individual with a brain lesion—*e.g.*, abscess—may be able to answer all questions intelligently, and to recognize the character and uses of any familiar object shown him and to give its name correctly, yet may be quite unable to read a single word of written or printed matter. He may be unable to recognize even the letters of the alphabet. This condition is called word-blindness (alexia), and is caused by a subcortical lesion which cuts off the centre for the memory pictures of written symbols from the other memory centres essential to coördinated speech. And, since the patient can not recall the form and appearance of the letters and written words, he is also unable to write. He may, however, be able to copy written matter, but does so with little or no understanding of the sense, or meaning, conveyed. This form of agraphia is called *sensory*, in contradistinction from the condition in which the patient recalls the forms of letters and written words, and can therefore read understandingly, but is quite unable to inaugurate the finger movements necessary in writing (motor agraphia).

Word-blindness and consequent sensory agraphia are determined by testing the patient's ability to read and write simple sentences in his mother tongue. The condition may occur independently or coexist with word-deafness.

Sensory Visual Aphasia (Psychical Blindness).—Again, if we suppose or find that our patient is unable either to name a familiar object shown him *or to bring to mind its uses*, but that he can recall its name when this, with others, is spoken for him, and from its sound can also recall its uses,—we have another variety of sensory aphasia dependent upon what is sometimes called psychical blindness. It is due to a subcortical lesion which practically isolates the special memory centre for visual impressions of objects seen. The "centre" is not actually destroyed, for the visual

memory pictures can be recalled by the sound of the name acting through certain indirect associational tracts. In such a case, we should not be surprised at finding the lesion rather far back,—*i.e.*, in the region of the second occipital convolution.

MOTOR APHASIA.—A somewhat kindred lesion, but producing quite different functional defects, is known as motor aphasia. This is due to a lesion directly involving the centre in which are stored the motor effort-memories necessary to the production of articulated speech (Broca's centre,—posterior part of third frontal gyrus). The patient can not talk, can not even repeat sentences word for word after another. He may, however, retain the use of a few monosyllabic words,—*e.g.*, yes, no, etc. This incapacity for speech may coincide with intelligent understanding of what is said,—this apparent anomaly being indicated by his gestures in response to questions, and possibly in some cases by the ability to convey his meaning in writing.

MOTOR AGRAPHIA, occurring alone,—*i.e.*, without disturbances of co-ordinated thought or speech,—is probably an exceedingly rare condition. For a typical case, we would have to suppose a lesion, cortical or sub-cortical, which had involved directly and solely, or at least cut off from association with other memory centres, the special centre for the motor effort-memories essential to the act of writing. The patient, while able to read printed or written matter with understanding, and while showing no evidences of apraxia,[2] is unable to write a single word. He can neither express his thoughts in writing, write at dictation, nor even copy the simplest sentence.

Motor agraphia and motor aphasia not infrequently coexist,—the effort-memory centre for writing being supposed to be not distant from the centre in which are stored the motor speech memories.

The differential points between motor agraphia (without motor aphasia) and sensory agraphia are repeated below:

Motor Agraphia.	*Sensory Agraphia.*
Due to a lesion which inhibits solely the mechanical act of writing; patient can therefore read with understanding.	Sensory agraphia is part of the condition known as word-blindness; patient cannot therefore either read or write.
Patient cannot write either at will or from a copy.	Patient cannot write at will, but usually can write from copy.

[2] Apraxia is a term used to describe the inability to think coherently which results from a loss of the concepts of familiar things.

CHAPTER XIII.

INTRACRANIAL LESIONS OF OTITIC ORIGIN: EXTRADURAL ABSCESS; PERISINOUS ABSCESS; SEPTIC SINUS THROMBOSIS OR PHLEBITIS; PURULENT LEPTOMENINGITIS; CEREBRAL ABSCESS; CEREBELLAR ABSCESS.

Avenues of Infection (Fig. 188).—Aside from the more favorable avenues of escape through the external cortex, it is clear that pus within the mastoid may travel in various directions, and in accordance with its

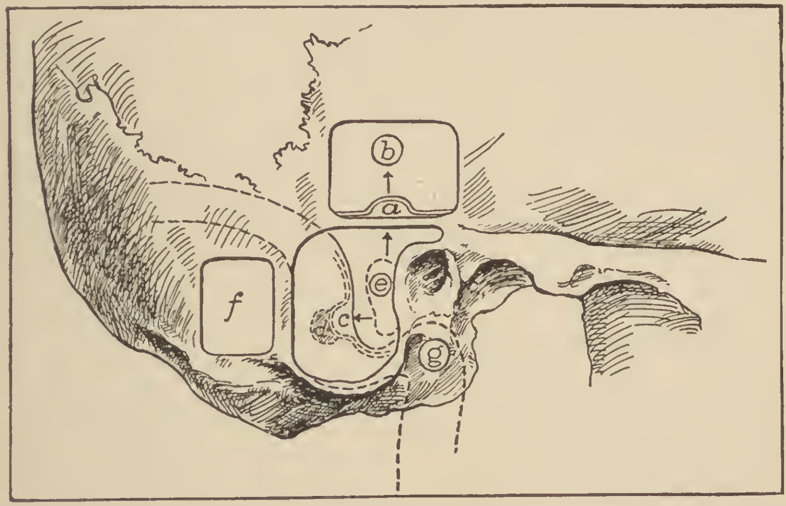

Fig. 188.—(Diagrammatic.) Otitic pathways of pus to brain or meninges. (*a*) Epidural abscesses; (*b*) temporosphenoidal abscess; (*c*) perisinous abscess; (*d*) sigmoid sinus thrombosis; (*e*) site of epidural abscess in contact with cerebellum; (*f*) convenient space for exposing cerebellar dura and which may represent nearest point for opening cerebellar abscess,—pus travelling from (*e*); (*g*) infection of jugular bulb, pus travelling downward from tympanum, or downward and forward from mastoid cells, or reaching bulb by the most usual route,—*i.e.*, from an infective lesion (*d*) in sigmoid sinus.

point of entrance may lead quite logically to different lesions within the cranium. Thus, it may perforate the roof, or tegmen antri, giving rise to an extradural abscess (a); or, as a later development of this lesion, to a cerebral abscess (b); or it may travel backward toward the posterior fossa of the skull, causing either a perisinous abscess (c), infective sigmoid sinus thrombosis or phlebitis (d); an extradural abscess in the neighborhood of the cerebellum (e), or a cerebellar abscess (f). Passing downward and forward from the mastoid, or downward from the tympanic cavity, the infection may spread directly to the jugular bulb (g), giving rise to a septic lesion within the jugular vein, not clinically distinguishable from a suppurative lesion within the lateral or sigmoid sinus.

It is obvious that germs entering the cranial cavity by any of the path-

ways above indicated may directly attack the meninges, giving rise either to a limited pachymeningitis or to a circumscribed or widespread purulent leptomeningitis. Fortunately, the latter condition is comparatively rare as a result of infection reaching the cranium by these routes, except as a complication of some suppurative lesion of the brain.

Still another very important pathway is furnished by the intermediate infection of the labyrinth. Pus from the infected labyrinth may reach the cranial cavity by three routes,—viz., (1) through the nerve channels into the internal auditory meatus, (2) from the cochlea through the aquæductus cochleæ, or (3) from the vestibule through the aquæductus vestibuli. Pus entering the cranium by any of these routes is particularly prone to cause widespread purulent leptomeningitis. Less frequently it causes cerebellar abscess or sigmoid sinus thrombosis. It must not be forgotten that pus reaching the cranium through the aquæductus cochleæ passes directly into the subarachnoid space.

EXTRADURAL ABSCESS; EPIDURAL ABSCESS.

The term extradural, or epidural, abscess is applied to any circumscribed collection of pus between the dural covering of the brain and the contiguous bone surface. It is the commonest of intracranial lesions of otitic origin. While not *per se* a dangerous condition, it is not without grave possibilities in the brain lesions to which it may give rise. Bezold believed that the presence of pus between dura and bone is a preliminary or intermediate stage of nearly every case of brain abscess.

Etiology.—The causes may be deduced from what has been said as to the possible avenues of infection. Great virulence of infection in a mastoid of pneumatic type, and in which the plates separating the mastoid and cranial cavities are thin, would seem to provide a favorable condition for the formation of these secondary collections of pus. On the other hand, a thick cortex, small pneumatic spaces, and pus under pressure would seem equally to favor their development. While depressed constitutional states should theoretically influence the rapid extension of bone necrosis, it has not been observed that old people or patients whose vitality is considerably below par develop this simplest complication of mastoid suppuration any oftener than the young or physically robust.

Extradural abscess occurs with far greater frequency in acute mastoiditis than with chronic suppurative otitis media with mastoid involvement. The lesion does occur, however, as a result of chronic middle-ear and mastoid suppuration. The discovery during the radical operation of an area of exposed dura is not particularly rare, and it is quite possible that this condition may represent in some cases an earlier collection of pus beneath the bone.

Symptoms.—The symptoms of epidural abscess may be *nil* or so indefinite as to be of little or no diagnostic value. The usual absence of characteristic symptoms, or our inability to interpret them correctly, is indicated by the fact that the lesion is rarely diagnosticated prior to operation for the relief of acute mastoiditis.

Among the symptoms which may be present are (a) severe localized pain, (b) severe headache, generalized or confined to the side of the lesion, and (c) elevation of temperature. These symptoms may, however, be wholly wanting, and, when present, their value is minimized by the fact that one or any of them may be present in severe uncomplicated mastoiditis. While it is an established fact that large extradural collections of pus occasionally give rise to focal or general symptoms of intracranial pressure, such symptoms are comparatively rare as a result of this lesion.

As physical signs we have sensitiveness to pressure over the site of the abscess—usually above the mastoid—and sensitiveness to light percussion over the mastoid region. These also are inconstant. The diagnosis is, therefore, usually dependent upon accidental opening of the abscess, or intentional exploratory uncovering of the dura on account of unusual severity of symptoms. Or, again, exposure of the dura may be made necessary by physical evidences of necrosis in the inner plate.

Extradural abscesses vary considerably in size, amounting in some cases to but a few drops of pus bathing the dural surface, in others reaching a very considerable amount confined under some pressure. In the case of large pus collections localized by surrounding adhesions between bone and dura, it is evident that the latter may be subjected to abnormal strain and tension. It is probably the latter condition which is chiefly responsible for the pain in certain cases.

Another physical variation which these cases exhibit is in the condition of the dura, which may be quite normal in appearance or may be covered with granulations,—this representing one stage of a localized pachymeningitis. Even in the presence of this latter condition, the symptoms are usually not sufficiently definite to form the basis of a diagnosis prior to operation.

The most common site of an otitic epidural abscess is between the tegmen antri and the dural covering of the temporal lobe. Next in frequency are the extradural spaces of the posterior fossa.

Prognosis.—In cases in which adequate surgical drainage is provided before the subdural space has become involved, the prognosis is distinctly favorable. The amount of pus present does not seem to influence the prognosis, the evacuation of a large abscess by careful but free removal of diseased bone being followed usually by convalescence quite as rapid as in uncomplicated mastoiditis. Nor does the presence of granulations upon the involved dural surface seem to have any unfavorable prognostic significance,—*i.e.*, always providing, of course, that the subdural space has escaped infection.

The comparative frequency with which this lesion is discovered during operations upon the mastoid, the frequent absence of any characteristic symptoms, and the usually uneventful and rapid recovery after surgical intervention, all point to a very important fact in the pathology of suppurative lesions of the temporal bone,—namely, the remarkable power of resistance to the action of infective micro-organisms inherent in the intact dura mater.

It has been intimated that a collection of pus in the posterior fossa

constitutes a far more serious lesion than a similar collection involving the dural covering of the cerebrum. This is in part due to the fact that a deep-seated abscess in contact with the cerebellar dura may perpetuate itself by spreading thence through the aquæductus vestibuli or auditory nerve channels to the labyrinth (Hinsberg, Bezold); but to a greater extent its gravity is due to the fact that pus collections in this location are far less likely to be discovered during operation upon the mastoid. A consideration of this latter fact led Jansen to suggest the wisdom of exploring this region as a matter of routine practice in the operation of mastoidectomy.

The treatment has already been indicated in speaking of the pathology of the disease. Careful removal of the diseased inner plate, or of the bone by which the pus is confined, is usually all that is required to convert the condition into one of simple uncomplicated mastoiditis. Usually the surgical treatment of extradural abscess forms part of an operation for the relief of suppurative mastoiditis. The technic will, therefore, be included in the description of the operation of mastoidectomy.

PERISINOUS ABSCESS AND INFECTIVE SINUS PHLEBITIS.

Perisinous abscess must in so considerable a proportion of cases be the immediate precursor of sinus thrombosis or phlebitis that there is every reason why the two conditions should be considered together. Only by such arrangement can the preventive treatment of the graver lesion—so far as prophylaxis is here possible—be duly emphasized.

Perisinous Abscess.—This term is applied to an extradural abscess involving the dural covering of the lateral or sigmoid sinus. In its commonest form, it is simply a collection of pus confined between the dural surface of the sigmoid sinus and the grooved or arched plate of bone which separates it from the interior of the mastoid process.

In its etiology and pathology a perisinous abscess does not differ materially from a similar (*i.e.*, extradural) abscess in other situations. There are, however, certain features of dural arrangement which render this particular lesion especially dangerous to the patient, unless promptly relieved by surgical means. These anatomical features have so practical a bearing upon the possible course of the disease as to call for brief description.

The dura covering the lateral convex surface of the brain is an exceedingly tough and resistant structure, very loosely adherent to the inner plate of the skull except along certain definite lines of attachment,—viz., along the lines of the principal sutures and at the edges of the groove for the lateral and sigmoid sinuses. In the region of the large venous sinuses, however, the dura divides into two layers, one of which lines the bone and forms the outer wall of the sinus, the other layer forming its inner wall. In the formation of the lateral sinus,—*i.e.*, from torcular to knee,—the inner layer of the dura lining the mid-cranial fossa joins with the inner layer of the dura lining the posterior fossa to inclose the sinus, and from this point they are continued inward as the tentorium cerebelli. In the formation of the sigmoid portion of the sinus, the outer layer of the dura is attached

rather firmly to the edges of the sigmoid groove, between which it lines the groove and forms the outer wall of the sinus. The inner layer is stretched rather tightly across the groove and thus forms the inner sinus wall.

The influence of the anatomical arrangement above described upon the possible course of a perisinous abscess must be obvious. Taking for purposes of comparison an extradural abscess in the mid-cranial fossa, it is clear that pus may spread widely between the parietal plate and the dura covering the temporal lobes, and meet with little or no resistance. On the other hand, pus between the dural surface of the sigmoid sinus and its bony capsule finds the following conditions: (1) Dura of only half the usual thickness, and in which any inflammatory process more quickly involves the inner coat. (2) More or less firm attachment of dura on either side to the edges of the sigmoid groove, pus being forced to travel toward either torcular or jugular bulb, or to cause marked compression of the outer wall. That such compression may be extreme is definitely proved by six cases recorded by Passau, in each of which the vessel was actually occluded, the sinus walls being in contact and circulation wholly cut off by the pressure of an extradural collection of pus.[1] (3) The inner dural wall being tense and inelastic, any degree of compression of the outer wall must tend to retard the venous flow and possibly increase friction,—either condition favoring the formation of a thrombus within the vessel. (4) Pus thus confined may cause softening of the sinus wall at any point, thus providing a pathway of infection leading to infective sinus phlebitis; or it may perforate simultaneously the two layers of dura at the point of their attachment to the edge of the sigmoid groove, carrying infection both to the sinus and to the cerebellum beneath (Figs. 189, 190, and 191). Possibly

FIG. 189.—Semi-diagrammatic picture showing section of sigmoid sinus and separation of dural layers.

FIG. 190.—Showing perisinous abscess, the thinness of the dural coat favoring perforation and intra-sinus infection.

FIG. 191.—Showing possible pathways of infection by simultaneous perforation of outer and inner coats, leading to infection both of sinus and cerebellum.

[1] Passau's Beitr., Bd. 3, Heft 1 and 2, 1910.

in this way may be explained the large number of cases seen or reported in which infective thrombosis and cerebellar abscess have apparently developed coincidently.

The author does not wish to convey the impression that he regards perisinous abscess as likely in most cases to lead quickly to the above very serious infections. As practical aural surgeons we know, from the many cases which have come under our personal observation, that a perisinous abscess may exist for a considerable period—may even produce a localized pachymeningitis, as evidenced by granulations covering the dura—without involving either the interior of the sinus or any subdural structure. With due consideration of these facts, we must not forget that the tendency of such a lesion, if not relieved by surgical means, must be to invade sooner or later either the sinus or the cerebellum, or both. It is of the greatest importance, therefore, that such an abscess be evacuated early,—*i.e.*, before it shall have given rise to a more serious lesion.

If we may draw any practical deductions from the above, they may be stated somewhat as follows:

(1) Whenever in a case of suppurative mastoiditis the symptoms have been such as to suggest the possibility of sinus infection, the sinus should be uncovered. In ordinarily skilful hands, this exposes the patient to no additional risk, and, even if no abscess be found, we shall at least have eliminated one source of danger.

(2) When in an operation for acute mastoiditis an unsuspected and unusual amount of pus is encountered,—*e.g.*, pus flooding the mastoid cells proper, the zygomatic cells, and possibly also the cells further back toward the occipital border,—it is safe and conservative surgery to expose the sigmoid sinus in order to eliminate the possibility of an abscess in this location.

(3) When physical changes are present leaving in doubt the health of the sinus groove, this bone should be removed. An exposed sinus is unquestionably a safer condition than a sinus in contact with bone the power of which to maintain its own nutrition is in doubt.

Symptoms.—The symptoms of uncomplicated perisinous abscess are so indefinite as to call for but brief mention. When the amount of pus between dura and sinus groove is small, there are usually absolutely no symptoms other than those which may occur with uncomplicated mastoid disease. With a large perisinous abscess there may be some evidences of sepsis simply as a result of the extended surface for pus absorption. Usually, however, there are no symptoms upon which one could possibly base a diagnosis of the lesion under discussion. Frankly, there is no chain of symptoms characteristic of uncomplicated perisinous abscess.

As with extradural abscesses in the mid-cranial fossa, perisinous abscesses are usually discovered during the course of a mastoid operation, either accidentally or through exploratory exposure for one or other of the reasons outlined above.

On removing the bone covering such a lesion, the conditions found may vary both as to the amount of pus and as to the state of the exposed dura. There may be, for example, only a few drops or a very considerable amount of pus,— the larger amount having no unfavorable significance as to the prognosis, provided the sinus wall is found to be healthy. The dura may appear quite normal even in the presence of an abscess of considerable size. On the other hand, it may show various physical signs of disease,— *e.g.*, it may be covered by granulations or may present the characteristic changes of superficial erosion. These structural changes of the dural wall do not, however, properly belong to this lesion, since they constitute rather the early changes in sinus phlebitis.

A point, however, which can not be too strongly insisted upon is this, that the presence of visible changes in the outer surface of the dura—*e.g.*, granulations, slight surface erosions, etc.—furnishes no proof that the vessel wall is diseased through its entire thickness or that the interior of the sinus has become infected.

Prognosis.—A perisinous abscess without noticeable changes of the dura, and which has received adequate surgical treatment, offers a perfectly favorable prognosis. Even in the presence of inflammatory changes in the sinus wall, the prognosis is usually favorable, unless there are accompanying symptoms of periodic septic absorption.

From what has been said we may deduce the chief indication of treatment,—*viz.*, the evacuation of pus by careful removal of the overlying bone. It is not sufficient to create a small opening through which pus may escape, the greatest safety being provided by a rather extensive uncovering of the sinus,—*i.e.*, certainly by a removal of bone coextensive with the size or extent of the abscess.

Granulations upon the dura are to be regarded as part of Nature's process of repair. Not on any account should they be curetted. On the contrary, the greatest care should be observed not to disturb or injure them, since a slight traumatism may convert a comparatively simple lesion into one of serious intra-sinus infection.

The surgical technic for exposing and draining a perisinous abscess forms part of the operative treatment of suspected sinus phlebitis, and will be described in detail in a later chapter.

Infective Sinus Phlebitis; Suppurative Thrombophlebitis of the Sigmoid or Lateral Sinus; Sigmoid Sinus Thrombosis.—The word phlebitis means literally an inflammation of a vein. Any inflammatory process —*e.g.*, the superficial changes so often discovered accidentally during mastoid surgery—should, therefore, fall under this heading. Practically, however, such a lesion causes recognizable symptoms only when the infection has involved the intima as well as the outer coats of the vessel. The term infective sinus phlebitis, being associated with a definite symptom complex, has come to apply in otology only to a lesion producing intra-sinus infection. And since an infective process involving the inner

coat of the lateral or sigmoid sinus very frequently gives rise to the formation of a thrombus at the site of the lesion, the terms, "infective sinus phlebitis," and "sinus thrombosis" were formerly regarded, and in otological parlance and literature were used, as practically synonymous. Of late years, however, we have come to recognize that the lesion resulting from intra-sinus infection may take the form of two pathologically distinct conditions, viz., infective clot formation (infective sinus thrombosis) and infective changes of the inner coat without clot formation (infective sinus phlebitis).

Etiology.—In the cases preceded by perisinous abscess, the causes are naturally in large degree those of the intermediate lesion. We have seen, however, that in many cases perisinous abscess does not give rise to intra-sinus infection. After an extradural effusion of pus has taken place, therefore, there must be certain factors which bear upon the progress of the lesion, and which determine whether it shall remain circumscribed or rapidly invade the sinus.

Probably changes in the individual s powers of resistance to disease furnish the determining factor in many cases. There can be no doubt, for example, that children suffering from suppurative middle-ear lesions complicating the acute infectious diseases more frequently develop intra-sinus infection than do children who have not been subjected to such severe systemic depletion. A very large percentage of the cases of infective sinus thrombosis which have come under the personal care of the writer have been among patients whose aural lesions have occurred either as a complication or sequela of severe constitutional disease.

The character of the infection is also a most important etiological factor in purulent sinus phlebitis. Leutert, in studying the bacteriology of cases of aural infection requiring operation, found in a large series that the streptococcus was responsible for all cases of infective sinus thrombosis. This view had the support of Bezold, who stated that in all cases of sinus infection examined in his hospital practice the streptococcus was found. Further rather positive corroboration of this conclusion is found in the careful studies of Dr. Libman in the Mt. Sinai Hospital of New York.

Pathology.—From the macroscopic appearances disclosed by surgical exposure of the sinus, it would seem that the morbid changes leading to infective sinus phlebitis may originate in two conditions,—viz., either (a) the presence of extradural pus,—*i.e.*, perisinous abscess; or (b) contact of dura with diseased bone. The latter may be assumed when no fluid pus is found between dura and bone, the two being simply soldered together through inflammatory adhesions. In either case the changes in the sinus wall probably follow each other in much the same order. Arguing an analogy between this lesion and similar processes elsewhere in the body we may assume that the dura in contact with diseased bone, or separated from it only by fluid pus, undergoes sooner or later the changes characteristic of an acute exudative inflammation; *i.e.*, first dilatation of the minute blood-vessels and subsequent retardation of the blood-current; next

transudation of serum and migration of leucocytes from the veins into the surrounding tissues. Unless quickly relieved, there is a proliferation of new cells, and the surface layer of endothelial cells undergoes necrosis. Upon this denuded surface granulations soon form. This probably represents a reparative stage of the lesion in which prompt and skilful surgical treatment results in the great majority of cases in fairly rapid resolution and recovery. The vessel wall is now thickened by the products of an acute exudative inflammation, but from further infection it is guarded by a protective layer of granulations. Removing from contact with these granulations all inflamed or necrotic bone, we pave the way for prompt recovery. Without such surgical relief, the lesion may progress in one of two ways,—viz., prolonged contact with the products of suppuration leads to necrotic softening of the vessel wall in its entire thickness at some point, thus providing a pathway of infection to the interior of the vessel; or the pressure upon the vessel wall by pus or by exuberant granulations may cause marked narrowing of its lumen at the point of the lesion, this leading to inflammatory changes of the intima. By whatever step the inner coat becomes involved in the inflammatory process, the mechanical result must be one or other of two conditions, viz., (1) the development of an intra-sinus erosion and clot at the site of the lesion (infective sinus thrombosis); or (2), in the absence of actual or demonstrable clot formation, the infection may involve and spread beneath the inner coat which, becoming eroded at one or several points, provides the means for periodic infection of the general blood stream (infective sinus phlebitis). It is essential that we recognize these two conditions as theoretically separate lesions in order (a) to explain the symptoms in those cases in which no clot is macroscopically demonstrable during operation; and (b) to give at least theoretic force to the contention of those surgeons who claim that resection of the jugular vein, rather than ligation, may in some cases be necessary and therefore is the safer routine procedure.

The first stages of clot formation may be accompanied by no clinical disturbances,—this being accounted for by the fact that the thrombus is probably always parietal at the start, and whatever germs it may imprison are at first imbedded within a firmly organized coagulum, and only reach the general current of the blood at a later priod when the clot shall have reached the stage of gradual disintegration.

Symptoms.—Considering under this head all conditions, subjective or objective, which may possibly throw light upon this lesion, it would seem logical to look for symptoms falling under the following heads:

(1) Symptoms due to periodic discharge of septic matter into the general circulation.

(2) Symptoms due to disturbance of cerebral circulation,—probable only in the case of a clot producing absolute obstruction. While complete occlusion is not particularly rare, definite symptoms, clearly attributable thereto, are.

(3) Bacteriæmia, or the demonstration of pus germs in the general blood current.

(4) Physical signs (often completely absent).

(5) Physical changes in the sinus wall, surgically exposed for inspection.

(6) Evidences of secondary foci of infection (abscesses) due to metastasis.

Symptoms of Periodic Septic Absorption.—There is probably no recognized lesion in which the chief clinical phenomena are more clearly the expression of the morbid changes present than in sinus thrombosis. As stated before, the formation of a firm parietal clot—*i.e.*, a clot adherent to the sinus wall and not obstructing its lumen—may produce absolutely no symptoms. Should such a clot be sterile,—*i.e.*, contain no pathogenic bacteria,—it may become organized into fibrous tissue, and never give rise to symptoms by which its presence might become known. How often this actually occurs, there is no means of determining. Presumably, however, most thrombi resulting from a suppurative process in contact with the outer sinus wall themselves contain the germ which has been the original source of infection. In this condition we may have a clot, peripherally sterile, but containing a central focus of infection, which finally leads to suppurative disintegration, with sudden propulsion of septic matter into the general blood stream. It is not surprising that the first definite symptoms are usually sudden, characteristic, and pronounced.

The symptom which first attracts the physician's attention is sudden and very marked rise of temperature. The temperature which has been hugging the normal line, or rising only slightly above it, suddenly shoots upward to 103° or even 105°F. Simultaneously the pulse-rate is increased, —*e.g.*, from 80 or 85 to 110 or 120 beats per minute in adults, or to a much higher rate of rapidity in children. During the period of pyrexia the patient is flushed and has the appearance of being exceedingly ill, as indeed he really is. During this stage he may complain of severe headache, this, however, in the writer's experience, being exceptional rather than the rule. The temperature, after maintaining its higher level for a few hours, suddenly begins to fall, and then drops rapidly to the normal line or a little above it. The fall of temperature is often accompanied by profuse sweating. He now enters upon an afebrile period of variable length, during which he feels comparatively well and comfortable. This sense of comparative ease and physical well-being is so commonly present and is in many cases so pronounced as to constitute one of the characteristic features of the disease. Unfortunately, this period of comfort and freedom from fever is distinctly limited. Usually in 24 hours, but in some cases not until two or three days have elapsed, the temperature again mounts rapidly to a point near or above the previous high level. The symptoms of the first febrile attack are now repeated, to subside rapidly after a few hours as the temperature again falls to the normal line. From this time the clinical course of the disease usually assumes a markedly periodic character, high fever and remission succeding each other often at fairly regular intervals.

In some cases before each rise of temperature the patient experiences a

pronounced *chill*, or rigor, which may be so pronounced as to be quite noticeable to nurses and attendants, and may last from five to ten minutes, or considerably longer. More often a distinct chill is not repeated, the patient complaining simply of a sense of chilliness just before the attack. In some cases neither a chill nor a sense of chilliness is complained of.

The blood count in septic sinus thrombosis, so far as has been observed, follows the same general laws which govern its changes in other suppurative lesions. The statement of Crockett,[2] that a gradual and progressive increase in the number of white cells to a point not usually exceeding 20,000 is a characteristic feature in septic sinus phlebitis, is of interest, and, if verified by further observation, will be of diagnostic value.

The cycle of septic phonomena above described undoubtedly forms the basis of one's diagnosis in the great majority of cases. It is clear that the two components of each febrile paroxysm are the logical results of the morbid process within the sinus,—the sharp rise of temperature marking entrance into the blood stream of septic matter from the disintegrating thrombus; the rapid fall to normal indicating their final elimination, or the final exhaustion of their toxic agents. In addition to the periodic character of the attack, the patient shows always a progressive loss of flesh, strength, and "tone," finally assuming the characteristic "septic look" which is so difficult to describe but usually so unmistakable to the practised eye.

The symptoms traceable to disturbed cerebral circulation are more often absent than present, and may be dealt with briefly. Crockett alludes to headache, nausea, and vomiting as among the occasional symptoms of sinus thrombosis. Headache of rather severe type may be present during the height of the febrile attack. Occurring during the period of remission, it is more likely to be due to the cerebral congestion caused by an obstructive clot. In two cases reported by Gruening[3] the sinus was absolutely occluded by an obstructive thrombus, and in each case the patient exhibited marked mental lethargy and drowsiness, one patient lapsing quickly into somnolence as soon as left undisturbed. Both of these patients suffered also from severe headache. In cases of parietal (*i.e.*, non-obstructive) thrombosis the above symptoms are absent.

A symptom which is occasionally present in obstructive sinus thrombosis is optic neuritis. Its occurrence has been observed and recorded by Gruening, Kipp, Crockett, Freidenberg, Jansen, and others. In Crockett's series of 60 operable cases, taken from the records of the Boston Charitable Eye and Ear Hospital, optic neuritis was present in 16, or slightly over 25 per cent. This is a larger percentage than has been recorded by other observers.

Hyperæmia of the optic nerve, optic neuritis, or choked disk, in a case presenting other clinical evidences of sinus phlebitis, would point rather

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[2] Crockett: Thrombosis of the Lateral Sinus, Annals of Otol., June, 1906.

[3] Gruening: Sinus Thrombosis of Otitic Origin and its Relation to Streptococcæmia, Annals of Otology, March, 1910.

strongly to an obstructive lesion,—*i.e.*, to a clot producing complete obstruction. Absence of all ocular changes is, however, absolutely without significance as an indication that the sinus is not diseased.

Most aural surgeons of large experience have seen cases of very extensive clot formation,—as, for instance, of a thrombus extending backward nearly to the torcular and downward so as to occlude the entire length of the jugular vein. In such cases it is easily conceivable that the thrombotic process may extend through the superior petrosal sinus to the cavernous sinus, practically obliterating the lumen of that vessel. Should this exceedingly rare complication occur, it would or might be announced by the following spectacular array of physical signs: œdema of the corresponding eyelids and about the brow, chemosis of the conjunctiva, exophthalmos and immobility of the eyeball from postorbital cellulitis.

Bacteriæmia.—It has long been known or believed that most of the important clinical phenomena of infective sinus thrombosis are directly attributable to the passage of germs from a disintegrating clot into the general blood stream. It is surprising, therefore, that attention was not directed earlier to the importance of blood cultures in cases of suspected sinus infection. Impetus has in recent years been given to the study of this question in America by the careful investigations of Dr. E. Libman[4] in the Pathological Laboratory of the Mt. Sinai Hospital of New York. The results of these investigations have corroborated the views expressed by Leutert[5] and Suepfle,[6]—viz., that the great majority of cases of sinus thrombosis are due to a streptococcus infection. Suepfle, from a careful analysis of a large series of cases, expressed his belief that cases of staphylococcus and pneumococcus infection rarely give rise to any intracranial complication. Libman, writing in 1908, after searching the literature of the subject, found an absence of authentic records of cases of sinus thrombosis due to the pneumococcus. These laboratory reports have the clinical support of Professor Bezold,[7] who stated in his text-book of otology, that all the cases of sinus thrombosis operated upon by him in his hospital practice were cases of streptococcus infection. A most interesting contribution to our clinical knowledge of the bacteriæmia of sinus thrombosis is found in a paper by Dr. Emil Gruening, published in the *New York Medical Journal* of June 5, 1909. This paper is based upon 10 cases of lateral sinus thrombosis operated upon by him in the Mt. Sinai Hospital and the results of the blood examinations by Dr. Libman. The blood for these cultures was taken from the median basilic vein. These examinations gave positive results in seven cases, being negative in the other three. "The micro-organisms causing the infection were in five cases the strep-

[4] Libman: Value of Bacteriological Examinations in Otology, with Special Reference to Blood Cultures, Arch. of Otol., xxxvii.

[5] Leutert: Bakteriologisch-Klinische Studien über Complicationen Acuter und Chronischer Mittelohreiterungen, Arch. f. Ohrenheil., Bd. 46, p. 190.

[6] Suepfle: Cited by Libman, ibid.

[7] Bezold: Text-book of Otology, p. 157.

tococcus pyogenes, in one case the streptococcus mucosus, in another the bacillus proteus. The same organisms had previously been found in the purulent discharge from the ear." Further proof of the practical value of blood examinations under certain circumstances is found in two cases, also reported by Dr. Gruening, in which, with no previous history of ear trouble, the sinus was explored solely on the strength of characteristic symptoms of septic absorption and a demonstrable streptococcæmia, an infected thrombus being found in each case.

A less positive conclusion as to the value of blood cultures as a means of determining septic sinus thrombosis seems deducible from the results published in the same year by Drs. Arthur B. Duel and Jonathan Wright, writing in collaboration.[8] Their report is based upon a series of 57 cases, treated in the wards of the Manhattan Eye and Ear Hospital, in which careful blood examinations were made. Of these 57 cases a positive bacteriæmia was found in 16. Of the 16 positive blood cultures, streptococci were present in 14, pneumococci in 2. Analyzing them further, according to the lesions present, we find that 4 were characteristic cases of septic sinus thrombosis, 1 of suppurative labyrinthitis with leptomeningitis, 2 were of frontal sinusitis, and the remaining 9 cases were of uncomplicated mastoiditis which made uneventful recoveries following simple mastoidectomy.

Clearly there are certain facts which should be thoroughly weighed and digested before arriving at a conclusion as to the surgical significance of a demonstrable streptococcæmia in cases of middle-ear suppuration. These facts may be marshalled somewhat in the following order:

Bacteriæmia is a usual accompaniment of septic endocarditis; it is not infrequently present in pneumonia and in purulent meningitis; it may be present in severe cases of tonsillar infection; it occurs not infrequently in the septic type of scarlet fever. It may, according to Duel and Wright, be present in cases of uncomplicated suppurative mastoiditis.

Libman's paper[9] is a perfectly fair and scientific statement of his results in an exceptionally brilliant series of cases. His investigations have helped to place at our disposal a very valuable aid in the diagnosis of doubtful cases. It does not appear, however, even when no other focus of infection can be located, that the mere presence of streptococci in the blood, unless reinforced by clinical manifestations of the disease, can be accepted as sufficient grounds for diagnosticating sinus thrombosis or for opening the sinus.

Physical Signs.—There are no constant physical signs of infective sinus thrombosis. In some cases of acute sinus phlebitis, the inflammatory process spreads backward along the mastoid vein to its point of entrance at the mastoid foramen, causing noticeable tenderness at this point. The position of this foramen varies somewhat, being usually about 1¼ to 1½

[8] Duel and Wright: Clinical and Pathological Significance of Bacteriæmia in Suppurative Otitis, N. Y. Med. Jour., Oct. 30, 1909.

[9] Libman: loc. cit.

inches behind the orifice of the cartilaginous meatus and on a level with its floor. Clearly defined sensitiveness to pressure at this point is regarded by some surgeons as a valuable diagnostic sign of sinus phlebitis. It is a condition which may easily be simulated by an extension of inflammation backward through a pneumatic mastoid to the large pneumatic spaces frequently present in this region.

Sensitiveness to pressure along the course of the jugular vein may be caused by an extension of inflammation to the jugular bulb and thence along the walls of the vein. When this inflammation is pronounced, the surrounding tissues may become involved, giving rise to a noticeable induration under the sternomastoid muscle. As a further extension of this secondary inflammation, the cervical glands lying in front of and behind the sternomastoid muscle may be distinctly enlarged.

The recognition by deep palpation of a cord-like mass supposed to represent a thrombus filling the jugular vein, has been mentioned by many authorities as a valuable physical sign in cases of extensive clot formation. Personally I have never been able to recognize it. One reason for my failure may be the fact that I have always believed that any forcible or deep palpation along the course of the vein in cases of suspected jugular thrombosis is an exceedingly unwise and dangerous procedure. It is conceivable that one might easily in this way dislodge and force into the general blood current a large portion of the infected clot, giving rise not only to pronounced systemic poisoning, but possibly also to disseminated metastatic foci of infection.

Moderate induration along the course of an infected vein, resulting from inflammatory changes in the surrounding tissues, is not very uncommon, and when present is an easily recognized and very valuable diagnostic sign. The writer is inclined to believe that this condition has in many cases been misinterpreted as representing an organized clot within the vein.

Macroscopic Changes in the Sinus Wall.—In a case of suspected sinus thrombosis, the exposure of the sinus for the purpose of inspecting its outer wall is not only justifiable, but clearly called for. Removal of the overlying bone may reveal one or more of the following conditions,—viz., (1) *Inflammatory adhesions between bone and dura.* This in some cases renders the uncovering of the sinus somewhat difficult and is an occasional cause of its accidental rupture. (2) *The presence of fluid pus in contact with an apparently healthy sinus.* This condition (perisinous abscess) is not particularly uncommon, and in adults may give rise to no symptoms whatever. In the case of young children, on the other hand, a perisinous abscess may cause a temperature of very septic type,—*i.e.*, sudden high rises alternating with remissions. I have seen a large number of such cases among children suffering from scarlet fever in the wards at the Willard Parker Hospital. The whole clinical picture may be such as to suggest intra-sinus infection. These symptoms usually subside after removal of the diseased bone,—unless, of course, there be a focus of infection within

the sinus. (3) *Presence of granulations upon the dura.* In addition to perisinous abscess, we frequently find the dura covered by a layer of granulations. With this condition the sinus wall may be very considerably thickened. Granulations do not form upon the normal dura. Their mere presence, therefore, is evidence of inflammatory changes involving at least the outer coats of the vessel. Such inflammatory changes with the superimposed granulations must necessarily mean thickening and increased resistance to pressure in that part of the sinus wall covered by granulations. In general, the increase in rigidity is proportional to the duration of the lesion. It does not necessarily follow, however, that the inner coat of the vessel is involved, and there is reason to believe that in a majority of cases such involvement does not take place if surgical intervention is not too long postponed. The point I wish to make is, that, wherever granulations are found, the sinus wall must be thickened and must offer increased resistance to the palpating finger. Unless the surgeon realizes this fact, he may receive the impression that he has under his finger a parietal clot and be led into the mistake of opening a sinus which Nature is guarding from infection, and which requires only the removal of superimposed diseased bone to insure a rapid recovery. (4) *Inflammatory changes with absence of granulations.* Conditions presenting even greater difficulties of interpretation are extensive inflammatory changes, occasionally encountered, with little or no tendency to the formation of protective granulations. Every aural surgeon of considerable experience will recall the anxiety which such cases have occasionally caused him. The sinus wall may present (a) a dull sodden, somewhat œdematous, or waterlogged appearance, the moderate lustre characteristic of normal dura being lost, and the usual bluish-gray color being changed to a somewhat purplish hue. The impression received is of an extensive pathologic change involving the entire thickness of the vessel wall. Or (b) the sinus wall may appear thinner than normally, and its normal lustre may have given place to an unnaturally glazed appearance. The macroscopic appearance is of marked atrophy, giving the impression of a tissue whose power of maintaining its own nutrition has been either destroyed or reduced to a minimum. When a sinus presenting this appearance conveys also a positive sense of resistance to the palpating finger, it is not surprising that the surgeon should suspect the presence of an adjacent clot. Again, (c) there may be areas of superficial erosion. This loss of surface epithelium may represent a traumatic result, marking the site of a former adhesion between the inflamed dura and the removed bone.

The writer at the present writing has under his care in one of the scarlet fever wards of the Willard Parker Hospital an adult patient whose sinus wall showed in different parts two of the conditions above described. The mastoid lesion was one of rapid and very extensive osseous necrosis. The sinus wall presented alternating areas of apparent atrophy and infiltration in different parts, and as a whole appeared so devitalized and extensively diseased that the writer predicted that the subsequent course of the lesion

would prove the interior of the vessel to have become infected. There had been, however, no symptoms altogether typical of septic poisoning through the sinus, and it was decided to keep the patient under observation for a day or two before subjecting him to further operative intervention. Since then ten days have elapsed, and no symptoms of further septic absorption have occurred. The diseased sinus is now covered with protective granulations and the patient is on the road to recovery.

It may seem from the above that the writer is devoting much space to a discussion of conditions having little bearing upon sinus thrombosis. That is not exactly the case. The physical conditions above described lead, if not surgically relieved, to infective sinus thrombosis. They form, therefore, a stage in the pathology of that lesion. The infective process may already have reached the inner coat of the vessel. We can not be sure, however, that intra-sinus infection has actually occurred unless, in addition to the physical changes above described, there are also some constitutional evidences of septic absorption.

As to the form of pathologic change in the sinus wall which may be accepted as positively indicating intra-sinus infection either present or inevitably to follow, my experience does not justify me in describing under this head any condition short of obvious gangrenous destruction of its vitality at some point. In short, while there undoubtedly are cases which justify the surgeon of large experience in opening the sinus upon the physical changes alone, the writer believes that they are exceptional. Certainly the surgeon whose experience with sinus lesions has not been large should not operate upon the sinus in the absence of characteristic symptoms of periodic sepsis.

Aspiration Movements. — Before leaving the discussion of physical changes, a word should be said of the so-called respiratory, or aspiration, movements occasionally seen in an exposed sinus.

The large veins of the neck, and notably the internal jugulars, show normally, if exposed surgically, very marked variations in calibre which occur synchronously with the respiratory movements. Thus, an exposed internal jugular balloons out during deep expiration and collapses during inspiration. These movements of the vein—exaggerated during forced respiratory efforts—are naturally less conspicuous during quiet respiration. Since the internal jugulars represent the direct continuation of the sigmoid sinuses, one would expect to observe similar movements of the latter when exposed during operation. Ordinarily, however, the sinus does not participate in these movements, sudden changes in its calibre being guarded against by various factors, prominent among which are the marked constrictions above and below the jugular bulb, and the sharp angular bends which obstruct the current in the neighborhood of the jugular foramen. When, however, the current is cut off by an obstruction (*e.g.*, clot) at the torcular end of the canal, the blood-pressure is so far reduced that noticeable collapsing of the sinus wall, synchronous with inspiration, occurs in that portion of the vessel between the obstruction and its exit

at the jugular foramen. Such a phenomenon in a case of suspected sinus thrombosis would, therefore, suggest an obstructing clot situated between the exposed sinus wall and the torcular. While its occasional occurrence and possible significance should be kept in mind, it is not a trustworthy sign, its unreliability being due mainly to two facts,—viz.: (1) If the obstruction were far back,—*i.e.*, posterior to the entrance of the superior petrosal sinus,—the blood from this sinus and from the mastoid emissary and posterior condyloid veins might be sufficient to maintain the usual pressure and prevent aspiration movements; and (2) with absolutely no clot or obstruction in any part of the sinus, moderate aspiration movements may be present in a perfectly normal vessel. In a case of mastoiditis operated upon some years ago by the writer, the wall of the exposed sinus showed not only well-marked pathologic change but also noticeable, almost flapping movements synchronous with the respiratory movements. This case, after causing the writer considerable anxiety, went on to uneventful recovery. This phenomenon in an unobstructed sinus I can account for only upon the hypothesis of an unusually large jugular foramen and a very large jugular vein which exerts during inspiration an unusually forcible drag upon the blood current in the sinus.

The course of the disease, after a focus of infection within the sinus has been established, is one of gradual, but ever progressive, loss of strength and vitality. The remarkable absence of symptoms, either subjective or objective, which usually characterizes the periods of remission during the early days of the attack, becomes gradually less marked as the patient becomes more and more septic. With the repeated entrance of septic emboli into the general blood stream, metastatic abscesses may be established in various parts of the body. I have seen such abscesses over the sternum, at the wrists, elbows, knees, or ankles. I have seen four or five such abscesses develop almost simultaneously in the same patient. In like manner, septic matter may find lodgement either in the lung or heart, giving rise to a septic pneumonia or to a septic endocarditis. In a case successfully operated upon by Dr. John B. Rae[10] for septic sinus thrombosis of the right side, the patient later developed symptoms of brain abscess in the left hemisphere, which was subsequently opened and drained. The cases cited indicate to some extent the multitude of dangers which hover about the patient suffering from this lesion, and which are ended only when the focus of infection within the sinus has been eliminated or its connection with the general circulation has been cut off. While the development of metastatic abscesses subjects the patient to intense suffering and increases enormously the drain upon his vitality, they do not render the prognosis hopeless. In one of the last cases operated upon by the writer, and in which he was obliged to resect the jugular vein, there was a large metastatic abscess involving the right elbow, which did not, however, interfere appreciably with the patient's rather rapid recovery. In a single

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[10] Rae: Transactions Am. Otol. Society, 1911.

case operated upon by Gruening,[11] metastatic abscesses developed and were successively evacuated in the right thigh, the right knee, the posterior aspect of each arm, the perineum, and last in the suprapubic region, the patient finally making a complete recovery.

Before making a diagnosis of sinus thrombosis, it is of course essential that all other diseases which might cause like symptoms should be definitely excluded. Among such may be mentioned pneumonia, typhoid fever, acute endocarditis, malaria, and certain cases of scarlatinal infection. The distinguishing features by which these conditions may be differentiated are known to all experienced physicians, errors in diagnosis being more likely to result from a too narrow conception of disease on the part of the aurist than from the absence of symptoms or physical signs which the trained diagnostician might be expected to detect. It is worthy of note that all the conditions referred to above are diseases in which a bacteriæmia is known occasionally to occur.

In the writer's experience, the cases presenting the greatest difficulties of diagnosis are those of children suffering from the septic type of scarlet fever. That such cases occur without any discoverable focus of localized infection is a fact well known to physicians in large practice among children. I have seen cases in which the temperature has for days run a course altogether characteristic of intra-sinus infection, but which have ultimately recovered without operation. With such facts in mind, the coincidence of severe scarlatinal infection and suppurative otitis media involves a problem of no little difficulty. At first thought, it would seem that such cases are pre-eminently of the class in which the examination of the blood from a distant vein for evidences of bacteriæmia would be of the greatest value. Unfortunately, the infectious diseases—and particularly scarlet fever—are known, even in the absence of sinus involvement, to be an occasional cause of streptococcæmia. Blood cultures are, therefore, not of very positive value in locating the principal focus of infection in cases of sepsis complicating the acute infectious diseases.

Prognosis.—We shall speak more intelligently of the prognosis if we accustom ourselves to recognize two distinct stages of the lesion,—viz.: (1) *suppurative sinus phlebitis externa*, characterized by inflammatory changes involving only the outer coats of the sinus wall; and (2) *infective sinus phlebitis interna*, or—since inflammation of the inner coat is usually coincident with clot formation—infective sinus thrombosis. Unless relieved by some form of surgical intervention, the prognosis in any stage of the disease is exceedingly unfavorable. Even when the inflammation is confined to the outer dural surface, it is difficult to conceive how resolution can take place, or intra-sinus infection be averted, so long as the dura is in contact with diseased or necrotic bone or bathed by confined pus. As soon, however, as we have removed all diseased structures from contact with the inflamed dura, we have paved the way for the processes of

[11] Gruening: Annals of Otol., March, 1910, pp. 148–151.

repair, and in most cases for fairly rapid recovery. In the great majority of cases intra-sinus infection does not subsequently take place.

In the second stage—*i.e.*, after a focus of infection within the sinus has been established—recovery without further operative intervention can not be expected.

Treatment.—The treatment of this lesion is purely surgical. That of the first stage—*i.e.*, before the interior of the sinus has become involved—is practically the same as that already advocated for perisinous abscess,—viz., the removal of all diseased bone from contact with the inflamed dura. In carrying out this provision, we have two surgical guides as to the extent to which the sinus should be uncovered,—viz., (1) until apparently normal dura is reached in both directions,— *i.e.*, toward the torcular and if possible toward the bulb; and (2) until the bone in contact with the sinus in all directions is perfectly sound and healthy. It is better to remove rather too much than too little, if by the latter we incur the risk of leaving unsound bone, or bone incapable of maintaining its own nutrition, in contact with the sinus wall. This, as a rule, is all that is required to bring about repair.

As soon as we are convinced that the interior of the sinus has become infected, there should be no hesitation or unnecessary delay in operating upon the sinus itself.

Indications for Sinus Operation.—The indications for opening the sinus are any conditions pointing with reasonable certainty to the presence of an infected clot. These, in the order of their importance, are:

(1) Mastoid disease plus symptoms of periodic septic absorption.

(2) Mastoid suppuration plus streptococcæmia.

(3) Changes in the sinus wall obviously gangrenous in character; only in exceptional cases, however, can the macroscopic evidences of disease be relied upon as evidence of intra-sinus infection.

I know that the views here expressed as to the limited value of macroscopic changes in the sinus wall as indicating intra-sinus infection will not have the support of some distinguished aural surgeons. The point in regard to which absolute unanimity of opinion may be assumed will usually not repay discussion. Those who advocate surgical intervention in cases of suspected sinus thrombosis in advance of symptoms of septic absorption, do so upon the hypothesis that the appearance of symptoms may express rather an advanced stage of the disease,—a stage in some cases too late for successful operative intervention. That symptoms of sepsis usually indicate clot disintegration is probably true; but that the beginning of clot disintegration represents in any considerable percentage of cases a stage in which the chance of successful intervention is materially reduced, I do not believe to be a fact.

The laboratory investigations of Dr. Libman, corroborated and checked by the clinical studies of Dr. Gruening, have brought out a fact of enormous clinical importance in the study of this disease,—viz., the very rapid disappearance of bacteria from the blood after resection of the jugular

vein. In most of their cases, the bacteriæmia was definitely checked within 24 to 48 hours after the ligation. In the light of this fact, it seems to the writer that we are more than ever justified in a reasonable delay in opening the sinus,—*i.e.*, until symptoms of systemic infection shall have demonstrated its necessity.

The accompanying illustration (Fig. 192) gives a clear and correct,

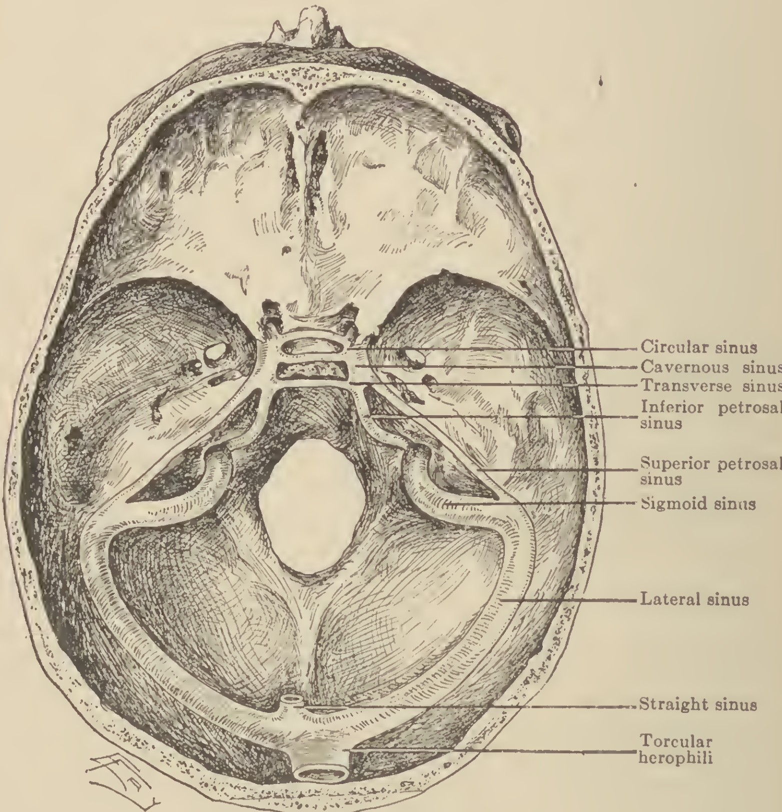

FIG. 192.—Venous channels of the brain.

though somewhat diagrammatic, picture of the large venous channels of the brain, and shows the relation, direct or indirect, between the sigmoid sinus and all the others.

Rationale of Operative Treatment.—The surgical treatment of infective sinus thrombosis has represented a gradual evolution, the successive stages of which may be traced in the following operations:

1. *Removal of Clot.*—The first operations were based upon the hypothesis that the clot itself was the chief source of systemic infection and that its removal would end the disease. The operation consisted in opening

the sigmoid sinus and examining its interior for the presence of an organized clot. The clot if found was removed. Sterile gauze was then placed against the wound in the sinus wall, which was allowed to heal. Its lumen was not obliterated. This operation resulted in a slight reduction of the mortality, but naturally failed in the great majority of cases.

2. *Removal of Clot and of the Diseased Sinus Wall.*—The disappointing results following the operation just described soon led to a recognition of its comparative uselessness so long as the diseased sinus wall was allowed to remain. The scope of the operation was therefore enlarged to include removal both of the clot and of the outer sinus wall throughout the extent of the incision,—*i.e.*, usually over a considerable portion of the vertical limb. This necessitated compression of the sinus above and below, and firm packing with sterile gauze of the intermediate portion, thus obliterating its lumen at the site of the operation. The results of this operation, while vastly better than those of that first described, yet failed to control the infection in a large percentage of cases.

3. *Removal of Clot and Diseased Sinus Wall plus Ligation of Jugular Vein.*—The latter operation marked the recognition of the rather frequent spread of the lesion from the sigmoid sinus to the jugular bulb or jugular vein below. It had become clear, that, for cases in which the limitation of the infective process to the sigmoid sinus could not be determined, the only treatment offering reasonable protection against further systemic poisoning would be one which not only attacks the initial lesion within the sinus, but also cuts off the connection of this vessel with the general blood stream by ligation or exsection of the jugular. The adoption of the jugular operation as part of the surgical treatment of intra-sinus infection has enormously reduced the mortality of the disease. Formerly regarded as among the nearly hopeless lesions, it is now looked upon as a disease in which the prognosis under skilful surgical treatment is fairly good.

The operations upon the sinus and upon the jugular vein will be described in detail in a later chapter.

ABSCESS OF THE BRAIN.

Etiology.—Etiologically we are concerned here with only one class of cases,—viz., those secondary to middle-ear or mastoid disease. While brain abscess may result from either acute or chronic suppurative otitis media, all investigations prove that the great majority of cases are due to the chronic form of the disease. Thus, Okada,[12] in a series of 145 recorded cases, found that 117, or 81 per cent., were due to chronic, while 28 cases, or 19 per cent., were caused by acute suppurative disease of the tympanum or mastoid. Neumann's[13] percentages, based upon an investigation of 151 cases, gave 88 per cent. as due to chronic and 12 per cent. as due to

[12] Okada: Klin. Vortr. aus d. Geb. der Otologie, etc., 3, p. 339.
[13] Neumann: Cerebellar Abscess, Eng. edition, p. 3.

acute disease. In Grunert's series[14] 91 per cent. resulted from chronic and 9 per cent. from acute tympanic or mastoid suppuration.

In line with the above are Jansen's statistics[15] compiled from the records of the Berlin Ear Clinic, in which 6 cases of brain abscess occurred in 2500 cases of chronic middle-ear suppuration, or 1 in 417, and only 1 in 2650 cases of acute suppurative otitis media.

Age.—All authorities agree that the great majority of victims are found in people between the ages of 10 and 30 years.

Sex.—Men are more frequent sufferers than women. Thus, of 204 cases collected by Körner,[16] 136 were men and 68 women. Of 188 cases collected by Neumann,[17] 127 were men and 61 were women.

Location.—The most frequent sites of brain abscess of otitic origin are the temporosphenoidal lobe and the cerebellum, the cerebral far outnumbering the cerebellar lesions. The relative frequency with which these two regions are attacked is perhaps best indicated in the following citations: Barr[18] in an analysis of 75 cases found the abscess to be in the temporal lobe in 55, in the cerebellum in 13, in both cerebrum and cerebellum (*i.e.*, two abscesses) in 4, in the pons in 2, and in the crus cerebri in 1.

Leaving out of our consideration abscesses in the rarer situations,—*e.g.*, in crus cerebri or pons,—and considering only those of the cerebrum and cerebellum, we have the following figures:

	Total number of cases.	Temporal lobe.	Cerebellum.
Barr[18]	68	55 (81 per cent.)	13 (19 per cent.)
Körner[19]	119	79 (67 " ")	40 (33 " ")
Heimann[20]	581	395 (68 " ")	186 (32 " ")
H. Tod[21]	100	65 (65 " ")	35 (35 " ")
Neumann[22]	532	336 (63 " ")	196 (37 " ")

In the above citation of Heimann's interesting statistics the total number, given here as 581, includes only cases in which a single abscess, situated either in temporal lobe or cerebellum, was present. In Heimann's paper the total number is increased to 645 by the inclusion of many cases in which double lesions—*i.e.*, abscesses in different parts of the brain—were present.

Brain abscesses, either cerebral or cerebellar, are usually single, but may be multiple. Abscesses may also coexist in the middle and posterior

[14] Grunert: Schwartze's Handbuch f. Ohrenheilk., Bd. ii, p. 849.
[15] Jansen: Berliner klinische Wochenschrift, 49, p. 1162.
[16] Körner: Die Otitischen Erkrankungen des Hirns, etc., 1896, p. 5.
[17] Neumann: ibid., p. 1.
[18] Barr: Diseases of the Ear, p. 288.
[19] Körner: Die Otitischen Erkrankungen des Hirns, etc., 1896, p. 103.
[20] Heimann: Arch. f. Ohrenheilk., 66, p. 257.
[21] H. Tod: Diseases of the Ear, p. 259.
[22] Neumann: Cerebellar Abscess, Eng. edition, p. 1.

fossæ,—*i.e.*, in the temporal lobe and cerebellum. Thus, in a series of 100 cases collected by Körner, coincident abscesses of the cerebrum and cerebellum were present in 6. In Hunter Tod's series of 100 cases from the records of the London Hospital the double lesion occurred 5 times, while Heimann in 645 cases found coincident temporal lobe and cerebellar abscesses only 10 times.

Bacteria.—The investigations of Leutert, Suepfle, Hasslauer, Libman, and others have abundantly demonstrated that the streptococcus more frequently attacks the brain during suppurative middle-ear disease than does any other one micro-organism. It does not follow, however, that the brain may not be in serious danger of invasion by other bacteria. That the contrary is the fact is made clear by the bacterial findings in 45 cases of brain abscess analyzed by Hasslauer.[23] Eleven of these were cases of infection following acute middle-ear suppuration, 34 complicating the chronic form of the disease. The following is a partial synopsis of Hasslauer's statistics:

Brain Abscesses complicating Acute Middle-ear Suppuration.

	Cases
Pus yielding streptococci in pure culture	6
Pus yielding diplococci in pure culture	1
Pus yielding staphylococci in pure culture	1
Pus yielding streptococci and diplococci (mixed infection)	1
Pus yielding streptococci and staphylococci (mixed infection)	1
Pus yielding diplococci and staphylococci (mixed infection)	1
	11

Brain Abscesses complicating Chronic Middle-ear Suppuration.

	Cases
Pus yielding streptococci in pure culture	10
Pus yielding streptococci, diplococci, and Gram-negative bacilli	1
Pus yielding streptococci, diplococci, and staphylococci	1
Pus yielding streptococci and bacilli	1
Pus yielding diplococci in pure culture	1
Pus yielding diplococci and bacilli pyocyanei	1
Pus yielding diplococci, streptococci, and b. proteus	1
Pus yielding diplococci and bacterium coli commune	1
Pus yielding diplococci and Gram-negative bacilli	1
Pus yielding diplococci and Gram-positive bacilli	1
Pus yielding staphylococci in pure culture	2
Pus yielding bacillus pyocyaneus in pure culture	1
Pus yielding typhoid bacillus in pure culture	1
Pus yielding tubercle bacillus in pure culture	1
Pus yielding pseudodiphtheria bacillus in pure culture	1
Pus yielding bacillus proteus in pure culture	1
Pus yielding bacterium coli commune and cocci	2
Pus yielding anaerobic bacilli in pure culture	1
Pus yielding anaerobic bacilli and streptococci	5
	34

[23] Hasslauer: Die Mikroöganismen bei den endokraniellen otogenen Komplikationen, Internat. Zentralbl. f. Ohrenheilk., Bd. v., Heft. I, pp. 15–16.

Considering together the cases caused by acute and chronic middle-ear or mastoid suppuration, we have the following results:

	Cases
Streptococci, in pure culture	16
Diplococci, in pure culture	2
Staphylococci, in pure culture	3
Bacilli pyocyanei, in pure culture	1
Typhoid bacilli, in pure culture	1
Tubercle bacilli, in pure culture	1
B. proteus, in pure culture	1
	25
Mixed infections	20
	45

An analysis of the above demonstrates clearly the following facts,—viz.: (1) That a very large proportion of all cases of brain abscess (in this series 16 out of 45, or 35½ per cent.) are due to a pure streptococcus infection. (2) There is no micro-organism capable of causing purulent otitis media which may not also give rise to brain abscess. (3) That in acute suppurative disease of the middle ear or mastoid there is greater danger of brain abscess in those cases depending upon a pure, or unmixed, infection than in those due to a mixed infection. Thus, of the 11 cases in this series 6 were due to an infection yielding streptococci in pure culture, 1 each to an unmixed diplococcus and staphylococcus infection, and only 3 to mixed infections. (4) Of cases of brain abscess secondary to chronic middle-ear suppuration the percentage of cases showing mixed infections is naturally much greater,—*e.g.*, 15 in 34 cases in Hasslauer's series. This does not signify that the average virulence of mixed infections is greater in chronic suppurative otitis media, but simply that the relative and actual frequency of mixed infections is greater; hence the greater number of brain abscesses from which more than one micro-organism may be obtained.

The above facts are for the most part in accord with the results obtained by other investigators. But, while considerable evidence has accumulated as to the relative potency of different bacteria in causing brain abscess, little is yet known as to certain differences in the character of the abscess itself as determined by the character of the invading micro-organism.

Neumann[24] believes that there is a distinct antithesis between the diplococci and certain bacilli,—particularly the Gram-negative anaerobic bacilli,—in the different structural changes to which they give rise in the brain. According to him, a characteristic property of the diplococci is their ability to produce an abundant secretion of fibrin from the blood of the brain substance immediately surrounding an inflammatory centre. Quickly following their invasion of the brain substance, there occurs, in

[24] Neumann: quoted by Körner, Die Otitischen Erkrankungen des Hirns, etc., 1908, pp. 56–57.

addition to the usual inflammatory reactions about the focus of infection, the formation of a more or less thick fibrinous membrane or reticulum, in the meshes of which large numbers of polynuclear leucocytes are deposited. This fibrinous membrane is gradually increased in thickness, its mechanical result being a tough, indurated wall separating the abscess cavity from the surrounding normal brain substance. In other words, it is a characteristic property of the diplococci, and to some extent of the cocci generally, to induce the formation of a firm and resisting abscess capsule,—a condition obviously influencing the prognosis favorably when a suitable pathway for the escape of pus has been provided. So strongly convinced is Neumann of this characteristic pathologic influence of the diplococci, that he regards their presence in pure culture, or their preponderance, in the pus obtained from a brain abscess as strong inferential proof of the existence of a well-defined abscess capsule.

The condition is quite different in cases of brain abscess due to infection by anaerobic Gram-negative bacilli. Such an abscess, according to Neumann, is characterized by the absence of a well-defined limiting wall or membrane, the boundary between abscess cavity and surrounding normal brain substance consisting chiefly of a layer of necrotic brain tissue or crumbling detritus. The pus from such an abscess is always markedly offensive, and, if some of the bacilli are isolated and cultivated, the same offensive odor is reproduced in the resulting growth. Ordinarily these micro-organisms exist as harmless saprophytes in the mouth, reaching the tympanic cavities through the Eustachian canals. Their occasional virulence in the ear and brain has not been explained. When either diplococci or Gram-negative bacilli are mixed with other bacteria, the controlling influence will depend upon the preponderance—either in numbers or in degree of virulence—of one or the other.

Pathology.—In a majority of cases the abscess is situated not far from the diseased area of bone forming the immediate gateway of intracranial infection. In others, constituting a decided minority, a considerable distance intervenes between the osseous lesion and the resulting abscess.

As to the process by which the infection reaches the interior of the brain, two modes of attack have been observed, viz.:

1. As a result of inflammatory changes in the tympanic or antral roof, —with or without perforation,—pus collects between the dura and bone in the mid-cranial fossa at a point corresponding to the necrotic area (extradural abscess). The dura, being in constant contact with pus, undergoes inflammatory changes, at first involving only its outer surface but later its entire thickness. Surgical evacuation of the pus not being provided, the inflamed dura undergoes necrosis at some point, thereby establishing a direct pathway of infection to the pia, and thence to the contiguous cerebral structure. This is probably the usual mode of infection in cases of cerebral abscess complicating acute tympanic or mastoid disease.

2. Without the intermediate formation of an extradural abscess, a beginning erosion in the bony plate covering the tympanic vault and antrum (tegmen tympani et antri) gives rise to inflammatory changes in the adjacent dura, the diseased dura and bone becoming adherent. The next stage of this process, according to Macewen,[25] is pronounced thickening of the dura with the formation of granulations upon its inner or cerebral surface. This results in a somewhat cone-shaped elevation (Fig. 193), which projects into and infects the pia mater. Throughout this raised area the necrotic bone, the meninges, and superimposed layer of brain matter are welded together by the products of inflammation. Lateral extension of this process being retarded by outlying inflammatory adhesions, the pathway of infection is toward the interior of the brain. This invasion may occur in three ways,—viz., (a) the blood-vessels and lymphatics of the pia may carry the infection to the adjacent brain structure, giving rise to a superficial abscess in close proximity to the area of osseous necrosis; (b) branches of the cerebral arteries, being for the most part terminal vessels with few anastomotic connections (Macewen), may become infected and lead to the formation of an abscess more deeply seated within the substance of the brain; or (c) disintegration at the centre of the inflamed area may provide a direct pathway for the passage of pus from the mastoid to the interior of the brain.

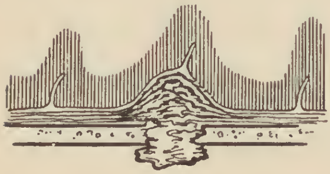

Fig. 193.—Invasion of brain through tympanic vault. (After Macewen.)

An abscess of the brain may vary in size from that of a pea to one measured by almost total displacement or destruction of the lobe in which it originated. This variation in size explains to some extent the varying clinical phenomena exhibited in different cases, and also the fact that an abscess may exist for a considerable period while producing absolutely no symptoms upon which a diagnosis could possibly be based.

It is now an accepted fact that brain abscesses differ histopathologically in accordance with the presence or absence of a limiting membrane or capsule. There are undoubtedly cases in which there is no apparent effort at encapsulation, the abscess focus being separated from the surrounding brain structure only by a thin layer of necrotic cerebral tissue (Neumann). More often, however, the suppurative invasion of the brain is followed by a productive or hyperplastic inflammation in the surrounding brain tissue, as a result of which a protective wall or capsule is gradually formed (Macewen, R. Müller). While such a wall or capsule undoubtedly delays the progress of the disease toward a fatal ending, the proportion of cases in which spontaneous healing through resorption and cicatrization occurs is probably exceedingly small.

Symptoms.—Considering the symptoms as they may occur from the

[25] Macewen: Pyogenic Diseases of the Brain, etc., pp. 71–72.

inception of the abscess to its successful evacuation or to the fatal ending, they may be divided into the following groups roughly typifying the stages of the disease,—*e.g.*, (1) those of the initial stage (onset), or stage of invasion; (2) those of the formative stage, or stage of gradual increase in intracranial pressure; (3) those of the latent stage, or stage of encapsulation; and (4) those of the terminal stage, corresponding to the final exhaustion of the patient through septic absorption, or to the rupture of the abscess into ventricle or subarachnoid space.

Initial Stage, or Stage of Invasion.—It is reasonable to suppose that breaking down of the anatomical barriers between the extradural and subarachnoid spaces — or, in other words, the sudden extension of the suppurative process to the pia mater — should give rise to definite symptoms. Macewen states that this stage of brain abscess formation is almost invariably announced by a chill, or rigor, and always by high temperature and severe headache. Vomiting, with or without nausea, is usually, but not invariably, present. These symptoms may be due to the initial changes at the site of the forming abscess cavity, but to the writer it seems much more probable that they are due solely or chiefly to a circumscribed cortical encephalitis surrounding the point of attack. Unfortunately, the onset, coming as it most frequently does during the course of a chronic suppurative otitis media, represents a stage which the aurist rarely actually sees. Its diagnostic significance may also be obscured by the number of other conditions which might cause very similar disturbances. These initial symptoms are usually of short duration, the fever and headache subsiding within a few days. They may leave the patient for a time in apparently fairly good condition, or may be immediately followed by symptoms of increased intracranial pressure.

Second Stage, or Period of Increasing Intracranial Pressure.—Between the onset and the development of further symptoms, a period may intervene in which no very characteristic phenomena are present. Following this intermediate period of quiescence, or in some cases crowding closely upon the onset, the first symptoms betokening the fully formed abscess are usually in some degree referable to increased intracranial pressure.

In order to take up the phenomena of the second stage in some regular order, it may be best to group them under three heads,—viz., (a) general symptoms of intracranial disturbance or compression, (b) symptoms due to septic absorption, and (c) focal symptoms.

The general symptoms of intracranial disturbance or compression include headache, restlessness and insomnia, temperature and pulse changes, mental lethargy or obscuration, inability to exert muscular force, changes in the eye grounds, gradually increasing somnolence.

Headache.—The headache of the second stage of brain abscess differs from that of the onset, or from that of acute leptomeningitis, in that it is not usually of the excruciating type which causes the patient to cry out. It has in typical cases rather the character of a dull, boring ache. It

has also this characteristic,—*i.e.*, that, while more or less constant, or at least very persistently recurrent, there is a tendency for its severity to be reduced as the abscess increases in size and the senses become dulled by increasing intracranial pressure. It varies considerably in its location in different cases, or it may attack at different times different parts of the head. Thus, it may be referred to the side of the head corresponding to the lesion, to the frontal region, or to the occiput; or it may alternate between the frontal and occipital regions. In most cases the headache varies in severity at different times, being usually worse at night. During the day it may be slight or absent,—or at least may not be complained of,—but as night approaches the pain returns or its intensity is increased, contributing to the prevention of sleep. With these patients, even during the period in which pain is not complained of, there is probably a more or less constant sense of cerebral disturbance or compression which must be inexpressibly harassing, and which probably contributes to the restlessness and insomnia which more or less characterize the disease.

Insomnia.—It seems a paradoxical statement that both somnolence and insomnia are strongly characteristic features of this disease. The patient may be practically sleepless during most of the twenty-four hours or, as is more often the case, his sleep is exceedingly fitful, disturbed, and easily broken, so that he sleeps and wakes many times within the hour, and in the morning is unrefreshed. When severe headache is present, the insomnia may of course be ascribed, in part at least, to that symptom. But even in cases in which headache is not particularly severe, insomnia is usually a prominent symptom. In such cases—*i.e.*, when headache is not complained of—the insomnia acquires special diagnostic importance from the fact that its degree and particularly its persistence are out of all proportion to any causes which can be discovered outside of a possible lesion within the skull. A patient who came under the writer's observation some years ago, and who died later from what proved to be the rupture of a temporosphenoidal abscess into the ventricles and subarachnoid space, described his insomnia somewhat as follows: "Though I am no longer able to work, I get very tired while doing nothing. I become not only very tired but very sleepy, so that I can hardly keep my eyes open; but when I lie down I can not sleep."

Temperature.—Following the initial rise described as characteristic of the onset, the temperature usually returns to normal or thereabout. From this time its variations may be confined within exceedingly narrow limits, not at any time rising to more than a point or two above the normal line. It is characteristic of a deep-seated brain abscess that the temperatrue may remain near the normal line, making occasional slight excursions either above or below it. Thus, in a case of suspected intracranial infection, a temperature curve ranging from a degree below normal to one or two degrees above it would be regarded as characteristic of brain abscess. On the other hand, in the presence of other reliable signs, a perfectly nor-

mal temperature could not be regarded as proof that the abscess was not present. Naturally, there are cases of brain abscess, even where no evidences of meningeal inflammation are present, in which the temperature either runs a higher course or makes occasional excursions to a higher level.

In cases presenting other conclusive evidences of brain abscess the presence or absence of fever would lead — so far as they might have any significance — to the following inferences: With complete absence of fever one would expect to find a rather deep-seated abscess, whereas persistent fever would point — in the absence of meningitis — to cortical irritation, and therefore to a lesion in close proximity to the cerebral cortex.

Presumably the occasional excursions below the normal point are due to pressure transmitted to some centre having to do with temperature control, the excursions in the opposite direction being the usual reaction to pus accumulation wherever it may be found in the body.

Pulse Changes.—With the gradual increase in intracranial pressure is frequently noted the characteristic slowing of the pulse-rate. The pulse, which under normal conditions has perhaps recorded 80 or 85 beats per minute, is gradually reduced to 75, 70, 60, 50, or perhaps 40 beats to the minute. This phenomenon is usually regarded as a result of increased pressure, the gradual reduction in pulse rapidity progressing with the gradual increase in the size of the abscess. On the other hand, quite marked pulse retardation has been observed in certain cases in which operation has given exit only to a very small amount of pus, and also in others ending fatally in which the autopsy has brought to light only a small abscess cavity. Probably the view held both by Macewen and Gowers—viz., that this symptom is due in some cases simply to irritation of some centre having an inhibitory influence over the heart action—is to be taken into account in explaining these differences.

When the abscess is successfully evacuated, the slow pulse-rate is usually promptly corrected, going first somewhat above normal—*e.g.*, 110 or 120 per minute—and then receding gradually to the normal line (Macewen).

Mental Dulness.—With increase in cerebral pressure, and depending therefore somewhat upon the size of the abscess, certain defects of cerebration usually make their appearance. The most characteristic mental change is one of mental lethargy. Even with unmistakable evidences of cerebral pressure, the patient is not exactly unintelligent, but may be decidedly dull mentally. In such cases, cerebration, though not ablated, is slow, difficult, and may be distinctly retarded. Thus, when asked a simple question, the answer, though correct, is given after an appreciable pause, as though its interpretation had required considerable effort. This retardation is sometimes so pronounced as to lead the physician to conclude that his question has not been heard. After he has ceased to expect a reply, however, the answer is given, and given correctly, showing that cerebration is obscured or impeded, but not otherwise disturbed. With

this condition the patient is often extremely drowsy, falling often into fitful sleep, from which he is easily aroused, but into which he quickly relapses when left undisturbed. Even more characteristic of a large temporosphenoidal abscess is the failure in the power of sustained attention. While the patient may be able to answer any simple question correctly, it may be quite impossible for him to concentrate his attention on any subject for more than a few minutes. For this reason it may be impossible for him to perceive the meaning of any but the simplest questions; or, having something to communicate, he may begin to speak and lose his train of thought while speaking; or, again, he may call for something to be brought to him, and altogether forget his request before it can be brought. Naturally, with mental processes depending upon such shallow and insecure impressions, they are not likely to be recalled after the patient recovers.

A striking example of the depression or obstruction without actual ablation of the mental functions is found in a case reported by Macewen[26] in which the patient recovered after evacuation of a large temporosphenoidal abscess. On the day of admission to hospital, he was able in reply to direct questions to give intelligent and correct replies as to his address, occupation, history of attack, his subjective symptoms, etc. On the day following evacuation of the abscess, however, he awoke without the slightest memory of the events of the previous day,—*i.e.*, "he realized for the first time that he was in a place unknown to him, and had no idea how he came to be in it."

Loss of Motor Will Power.—With gradual loss of the power of sustained attention, there is always an increasing tendency toward drowsiness. The condition is one of mental and physical lethargy and indifference. The patient can not be stimulated to the exercise of any sustained effort, either mental or physical. While paralytic disturbances may be completely absent, he presents in advanced stages of the disease an appearance of marked physical weakness, which is due not to actual loss of muscular force, but to loss of the potential will power necessary to call it into activity.

Eye-ground Changes.—Changes in the eye-grounds—*e.g.*, papillitis, optic neuritis—may occur with any intracranial disease causing increased pressure. They may be present, therefore, in cases of brain tumor, brain abscess, or with meningitis.

Urbantschitsch[27] cites the work of E. and O. Ruttin, who made systematic examination of the eye-grounds in a large number of intracranial lesions coming under observation in the Urbantschitsch clinic and hospital service in Vienna. According to these investigators, the eye-grounds were found normal in all cases of otitic abscess of the temporal lobe; they were normal in the majority of cases of cerebellar abscess, whereas in

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[26] Macewen: Pyogenic Diseases of the Brain, etc., pp. 155–161.
[27] Urbantschitsch: Lehrbuch d. Ohrenheilk., p. 514.

advanced otitic meningitis optic neuritis was commonly present. Arguing from these premises they concluded that a positive finding of papillitis or optic neuritis in a case of suspected intracranial involvement is to be regarded as pointing strongly away from otitic cerebral abscess as the lesion present. Interesting as these observations may be, eye-ground changes resulting from brain abscess have been too frequently recorded to warrant so sweeping a deduction without further investigation.

So far as we are justified in any positive statement, the consensus of opinion is probably about as follows: Eye-ground changes (papillitis, choked disk, optic neuritis) occur much more frequently as a result of slow-growing brain tumors than with brain abscess. They also occur more frequently with meningitis than with brain abscess. They do, however, result from brain abscess in certain cases.

Papillitis is more frequently seen in brain abscess than is optic neuritis, for the reason that the rapid course of a cerebral abscess either to a fatal termination or to recovery through operation often does not allow time for the development of a well-marked neuritis. When present, optic neuritis is usually a late sign, coming only when the abscess has reached considerable size and produced very marked increase of intracranial pressure.

Macewen[28] states that optic neuritis occurs "neither in cases of cerebral abscess which have run a rapid course, the period for its production being too short, nor where the cerebral abscess is small and the degree of surrounding inflammatory reaction is slight." If this be true, the association of optic neuritis with other well-marked symptoms of brain abscess should be of favorable import to this extent, —viz., that it would seem to point to a slowly developing abscess of considerable size, and one having presumably a well-formed capsule. Such an abscess should offer favorable conditions for successful surgical evacuation.

Papillitis or optic neuritis may occur in one or both eyes, and may be confined to either eye. That is to say, an abscess in the right temporal lobe may give rise to fundus changes in the left eye, or *vice versa*. They are in no sense, therefore, focal or localizing symptoms.

To epitomize: Changes in the eye-grounds usually mean somewhat prolonged increase of intracranial pressure. Associated with other symptoms of brain abscess, they are undoubtedly an aid to diagnosis. Absolutely normal eye-grounds do not, however, constitute a disproof of such a lesion.

Vomiting, which is spoken of in most text-books as an occasional symptom of cerebral abscess, is undoubtedly a common feature of the onset. After the initial stage is past, vomiting is comparatively rare. As a late symptom it is a much more frequent accompaniment of cerebellar abscess than of abscess of temporal lobe. Occurring late in temporo-

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[28] Macewen: Pyogenic Diseases of the Brain, etc., p. 141.

sphenoidal abscess, it would suggest at least the possibility of meningeal involvement.

Symptoms of Sepsis.—The symptoms of sepsis in this disease differ somewhat from those which betoken septic absorption from suppurative lesions in other parts of the body. The temperature curve, for example, usually does not show the wide fluctuations commonly associated with septic absorption; on the contrary, its excursions are usually confined between narrow limits, and it may remain during considerable periods practically normal. As offsetting this paradoxical absence of fever, there are two conditions which probably in many cases have to do with the septic condition of the patient,—viz.:

1. There is a progressive, and in some cases remarkably rapid, loss of flesh and strength, which no lesion of otitic origin short of intracranial infection could possibly give rise to. Meningitis being excluded, the rapid progression of this change, watched and measured from day to day, may of itself constitute a strong indication of cerebral abscess.

2. Another feature which is not easily described in exact or scientific terms, but which is sometimes most striking, is the early disproportionate appearance of grave illness as compared with any other symptoms present. This contrast is in some cases so striking as to suggest at once an intracranial complication.

Probably these changes in the weight, strength, and general tone are due in part to disturbance of certain centres having to do with tissue metabolism, and in part to the peculiar potency of septic agents absorbed through the structures of the brain. That they are not to be regarded altogether as trophic changes due to compression, seems deducible from their absence in certain cases of encapsulated abscess of considerable size,—so-called ambulatory cases,—in which the patient's condition is fairly well maintained until shortly before the terminal stage is reached.

Focal Symptoms.—Under this heading we may for convenience include any clinical change, subjective or objective, which throws light upon the site of the lesion.

Muscular Paresis.—In large temporal lobe abscesses, even when there are no phenomena which indicate the particular gyri involved, there is apt to be a slight contralateral paresis of the upper extremity. This paresis may be so slight as to escape notice unless carefully looked for. On the other hand, a careful observer may note a comparative languor, or effort, in the movements of the hand and arm opposite to the lesion as compared to its fellow. The best means of determining this unilateral deterioration in muscular power is by comparing the grip of the two hands, either by requiring the patient to squeeze the examiner's hand, first with one hand and then the other, or, better still, by means of a dynamometer. The paresis of the hand and arm is noticeably present in many cases in which no loss of power in the lower limb can be determined. The relative frequency of this involvement of the upper extremity and the comparative immunity of the lower, is made clear when we consider the relation of an abscess in the

temporosphenoidal lobe to the motor area above it; for, if we regard this paresis as resulting wholly from transmitted pressure, it becomes evident that this pressure will in a majority of cases be exerted chiefly upon the motor area of the face, shoulder, arm, and hand, and only slightly or not at all upon that of the leg or foot (Fig. 194).

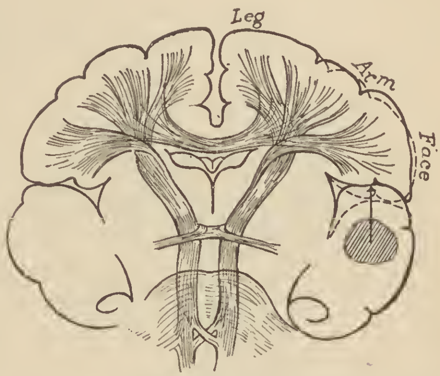

Fig. 194. — (Diagrammatic.) Vertical cross section of brain, showing influence of transmitted pressure from temporosphenoidal abscess (shaded area) upon motor area above.

Muscular Spasms.— Muscular contractions are by several authors mentioned as among the occasional symptoms of brain abscess. They can occur as a result of such a lesion only when, directly or indirectly, the motor area is irritated. A large and rapidly formed abscess in the temporal lobe might induce localized convulsions by rapidly developed pressure upon the lower part of the precentral lobe, but the effect of such pressure is much more likely to be the reverse,—*i.e.*, in the direction of paresis or paralysis of the muscle groups involved. A class of cases in which muscular contractions would be more likely to occur are those described by Neumann in which there is little or no tendency to the formation of a limiting capsule. This absence of a limiting membrane might result in a surrounding encephalitis which might easily extend cortically from the temporal lobe to the motor area of the frontal. Definite muscular spasms are exceedingly rare as a symptom of temporosphenoidal abscess. Should they occur without symptoms of meningeal involvement, they would, of course, have some value as throwing light upon the position and particularly upon the size of the abscess, or as betokening an accompanying and extending encephalitis.

Localized Paralyses.—The theory of contralateral paralysis as a result of cortical brain lesions is so universally understood as hardly to require mention in a modern text-book. From the usual position of otitic abscess, —*i.e.*, in the temporosphenoidal lobe,—monoplegias are rarely seen. If they did occur they would suggest an extension of the suppurative process from one lobe to another, as it is hardly conceivable that the influence of pressure transmitted from the temporal lobe to the precentral cortex should be limited to the motor area of a single muscle, or to its closely related group. A true monoplegia would, therefore, suggest very strongly the possibility of a secondary abscess.

More or less widespread paralysis is an occasional phenomenon of otitic brain abscess, and may result either from transmitted pressure upon the cortical motor area or from direct involvement of the internal capsule. As to which type of lesion exists in a given case, we may take

into consideration the following conditions, shown diagrammatically in Figs. 195 and 195a: (1) A temporosphenoidal abscess capable of producing widespread paralysis by pressure upon the motor cortex would necessarily be of a very considerable size; it would be likely therefore to produce other manifestations of increased pressure (*e.g.*, mental dulness or obscuration, slow pulse, and possibly, in a left-sided lesion, some form of aphasia).

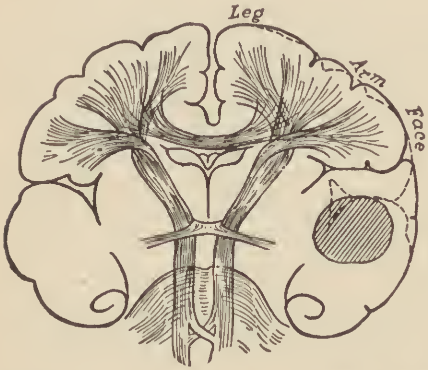

Fig. 195.—(Diagrammatic.) Vertical cross section of brain, showing a large temporosphenoidal abscess (shaded area).

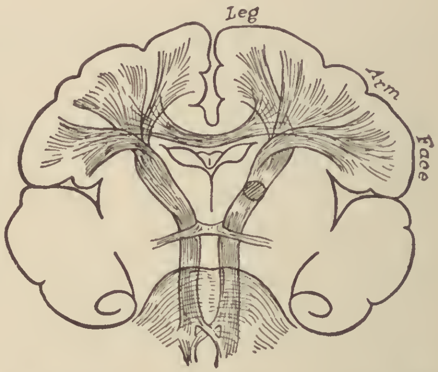

Fig. 195a.—(Diagrammatic.) Vertical cross section of brain, showing small abscess (shaded area) involving internal capsule.

From the anatomical relation of the temporal lobe to the ascending frontal lobe, it is clear that pressure would be exerted most forcibly upon the lower third (face, arm, etc.) and least on the upper third (foot, leg). This is shown by Fig. 195. (2) An otitic abscess of comparatively small size involving the internal capsule would be (a) more likely to produce widespread hemiplegia, and (b) less likely to give rise to other pronounced pressure symptoms (Fig. 195a).

The presence, therefore, of contralateral paralysis, pronounced in face, arm, and hand, but slight or absent in the lower limb; markedly slow pulse; pronounced mental dulness, drowsiness, etc., would suggest the results of pressure from a large temporosphenoidal abscess. On the other hand, a widespread paralysis or even paresis,—*i.e.*, involving the leg and foot equally with the upper extremity,—particularly if other pressure symptoms were absent or inconspicuous, would point to a lesion involving the internal capsule.

A form of paralysis which has been repeatedly described as resulting from brain abscess is that in which a contralateral hemiplegia coexists with motor oculi paralysis (ptosis, external squint, inability to rotate the eye in other directions) on the side corresponding to the lesion. This may be produced in two ways,—*i.e.*, by a lesion involving the crus cerebri (explained on pages 329-330), and by a large temporosphenoidal abscess exerting pressure at once upon the cortical motor area and on the third nerve. Macewen has described two such cases resulting from direct pres-

sure upon the motor cortex, and he gives the following differential points between the two lesions: When the contralateral paralysis is the result of pressure transmitted to the cortical motor area, the foot and leg, from the position of their motor centre, escape wholly or are only slightly involved. When caused by a lesion of the crus cerebri, the hemiplegia is usually widespread,—*i.e.*, involves equally the upper and lower limbs.

Pupillary changes may or may not be present in cases of temporosphenoidal abscess, depending, no doubt, on the location or size, and the consequent disturbance of pressure. When present, they differ from changes in the fundus oculi in that, while the latter may involve either eye, or both, pupillary changes occur, as a rule, only in the eye corresponding to the side of the brain in which the abscess is located. It is, therefore, to some extent a focal symptom. The pupil may be either contracted or dilated, or, beginning with contraction, may end—as the abscess increases in size and intracranial pressure is augmented—in stable mydriasis (Macewen). In some cases in which the pupils are apparently of equal size, a useful and interesting sign of abnormal unilateral pressure may be found in the sluggish pupillary reaction of the eye corresponding to the side of the lesion, which responds slowly to accommodation and light. In such a case one would expect stable inequalities of pupillary contraction to develop later, unless of course the abscess were evacuated by surgical means.

Aphasia.—The various cortical centres in which are stored the memory pictures, or impressions, essential to coherent speech are situated in the left side of the brain in right-handed persons and in the right hemisphere in left-handed persons. Aphasia may be looked for, therefore, only in cases of suspected disease in the side of the brain in which these centres are known to be,—*i.e.*, the left brain in a right-handed individual, and *vice versa*.

NOTE.—As the subject of aphasia is a most important one and one to which very limited space can here be given, the student is referred to Chapter XII, in which will be found a chart of the more important memory centres concerned in speech, with a brief discussion of their functions and the various speech defects following their injury or destruction.

Sensory Aphasia.—The commonest speech defect in otitic abscess is the form of sensory aphasia known as *word-deafness*. Its presence is determined in the following way: The patient is shown some familiar object—*e.g.*, a watch—and is asked to name it. He may be unable to do so, or may call it by some name having no relation to a time-recording instrument. He is next asked what it is used for, and he answers correctly "to tell the time of day with." We know, therefore, that he recognizes its form, and from this its uses. We now ask him the uses of some familiar object which is not exposed to his view,—*e.g.*, a saw, a hammer, or a pencil,—and if the name awakens no memory of it, but he is able to recognize it as soon as it is shown to him, we have a clear example of word-deafness. Now repeating our first experiment of showing him a watch,—of which he recognizes the uses but can not recall the name,—ask him if it is a knife, a hammer, or a

watch, and he may promptly say that it is a watch. This shows that his aphasia is due to a subcortical lesion which has cut off the memory centre for sound impressions from association with other memory centres concerned in speech, but that the "centre" itself is not destroyed, and that its activity can be temporarily re-established through certain association tracts when the object is seen and its name heard simultaneously. It is, therefore, a partial word-deafness, as distinguished from the total word-deafness which would occur if the cortical centre for sound memories were actually destroyed.

Partial word-deafness resulting from a subcortical lesion gives rise to the commonest form of sensory aphasia met with in otitic brain abscess, a fact which might be expected from the position of the special centre involved,—a position naturally exposed to pressure from a temporosphenoidal abscess (Fig. 187). This form of sensory aphasia may vary somewhat in different cases in accordance with the extent to which the centre for sound memories is isolated. Thus we may have: (a) The common form, described above, in which the patient, though mentally depressed and disinclined to any unnecessary speech, is yet able to communicate his wants in short, simple sentences. The partial word-deafness of such a patient might be easily overlooked, unless he were subjected to systematic tests by the method above described. (b) In cases of more advanced word-deafness the loss of memory for word sounds may be so pronounced as to be easily noticed in his simplest utterances. Such an example may be found in a very interesting case of left temporal lobe abscess reported by McKernon,[29] in which the patient in endeavoring to communicate his wants would both "mispronounce words and call articles by wrong names." (c) Still another phase of word-deafness is found in the case reported by Gehuchten and Goris,[30] in which the patient, a man of 40 years, suffering from left temporosphenoidal abscess, had so far lost all sound memories that he could not understand the simplest spoken sentence. That the disability was due simply to the isolation of the centre for sound memories was shown by his ability to understand written communications perfectly and to answer all questions intelligently in writing.

A moment's thought will show that the above differences are apparent rather than real, and that they represent only different grades of partial word-deafness. A very interesting case of sensory aphasia belonging to the same class, and one which well illustrates the fact that the aphasic individual may within certain limits think fairly coherently, was recorded by Dean, of Iowa.[31] This patient, a man of 35 and an habitual smoker, developed an otitic abscess in the left temporal lobe, the symptoms of which were temporarily relieved by operation. This man was able to recall the symptoms he had experienced before surgical relief was obtained,

[29] McKernon: Report of a Case of Temporosphenoidal Abscess, Annals of Otology, May, 1902.

[30] Gehuchten and Goris: Le Névraxe, vol. iii, p. 65.

[31] Dean: Brain Abscess of Otitic Origin, Annals of Otology, vol. xix, p. 543.

and he defined very clearly the type of aphasia which had been present by his account of the suffering he had endured for want of tobacco. He wanted to smoke and knew what he wanted, but stated that he had been unable to make his wants known, for the reason that he could not command any of the words required to indicate tobacco or its uses.

Psychical blindness (sensory visual aphasia) in pure form is comparatively rare as a result of otitic brain abscess. As with word-deafness, the patient suffering from psychical blindness is unable to give the name of a familiar object shown him. His inability to do so is due, however, not to loss of the memory pictures of word sounds, *but to a loss of memory for the visual impressions of familiar things.* He can not, therefore, give the name of a hammer, knife, or pencil shown him for the reason that these objects no longer appear familiar to him, and, therefore, awaken no memory either of their uses or their names. The condition is due to a lesion—usually subcortical—which has cut off from association with other centres the special memory centre for the visual impressions of things. This centre, it will be remembered, is situated in the second occipital convolution (Fig. 187). The aphasia dependent upon psychical blindness is clinically distinguishable from that associated with word-deafness by the fact that the patient is able to recall the uses and general character of familiar objects when their names are called, though he may receive no mental impressions from a familiar object seen.

Word-blindness (causing sensory agraphia) may coexist with psychical blindness or may occur independently. It is present when the patient can no longer recall or recognize the forms either of individual letters or written words. He is, therefore, unable either to read or write (sensory agraphia). It is due to a lesion which isolates the centre for the visual memory pictures of written words or symbols, this centre being situated in the angular gyrus.

Motor aphasia is mentioned in many text-books as among the occasional symptoms of brain abscess. It would occur with any lesion which destroyed or isolated the memory centre for the motor efforts necessary in speaking, this being located in the posterior end of the third frontal convolution, "Broca's convolution." As an example of motor aphasia may be mentioned Banti's case[32] in which the patient, while mentally quite coherent and understanding everything that was said to him, could not utter a single word. Though unable to speak, he had a clear understanding both of spoken and written language, and could himself communicate his thoughts quite rationally by writing. This case finally ended fatally, and the autopsy brought to light "a patch of yellow softening at the posterior end of the third frontal convolution."

Many cases of motor aphasia resulting from brain tumor and other lesions have been recorded, but I have personally never known of a case secondary to otitic brain abscess. Nor will it be found easy to find a well-

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[32] Banti: Gordinier, Anatomy of the Central Nervous System, p. 473.

authenticated case recorded in otological literature. It is mentioned simply as a possible, but certainly rare, symptom of otitic abscess.

Motor agraphia (described on page 337) is dependent upon a somewhat analogous condition, involving, however, the centre for the motor effort memories involved in writing. According to Gordinier, who described a classical case coming under his own observation,[33] this centre is situated in the posterior end of the second frontal convolution. It rarely or never occurs singly in brain abscess secondary to otitic disease.

Course of the Disease.—Brain abscess, unless relieved by surgical means, leads regularly, either through a more or less prolonged latent period or more rapidly, to the terminal stage ending in death. Of the latent stage, or stage of encapsulation, we know nothing beyond the fact that, in certain cases in which death has resulted from other causes, the autopsy has revealed either the cicatrices representing small abscesses long since absorbed or, more rarely, an encapsulated cyst or abscess which had long ceased to cause any characteristic symptoms. Such spontaneous cure or prolonged encapsulation without untoward result is probably exceedingly rare.

The Terminal Stage.—In fatal cases death usually comes in one of two ways,—*i.e.:*

(a) The progressive loss of strength and flesh leads finally to extreme emaciation, the patient presenting a mere shadow of his former self. The mental apathy or dulness which characterizes the earlier stage is gradually deepened into a condition of somnolence from which it is difficult even momentarily to arouse him, this in turn giving way to a condition of coma. The pulse, which has perhaps been abnormally slow, may become rapid and irregular. The respirations, on the other hand, are usually slower, sighing, or may become stertorous in character. Death finally results from exhaustion.

(b) In other cases death results from rupture of the abscess into the ventricles and subarachnoid space. This may occur only when the patient is near death from exhaustion, or it may occur comparatively early in the disease. I recall one instance in which an unsuspected abscess ruptured while the patient was up and walking about the ward. In either case the catastrophe is invariably followed by a characteristic train of symptoms. The face assumes a death-like or livid hue. The temperature quickly rises to 103° F., or higher, while the pulse-rate, which may have been abnormally slow, is increased to 140 or more per minute. The respirations become slower, labored, and finally stertorous, as the patient sinks into a stuporous condition. The pupils may be inactive and unequally dilated. It is obvious that the flooding of the brain surfaces with pus must give rise to more or less diffuse suppurative leptomeningitis, this condition being largely responsible for the quick termination. Death usually ensues within one or two days.

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[33] Gordinier: Amer. Journal of Med. Sciences, May, 1899.

Prognosis.—Leaving out of our consideration the exceptional instances of spontaneous recovery, it may be said that the usual termination of a brain abscess which has not been relieved surgically is death. The treatment, therefore, is surgical, and will be described in a later chapter dealing with the operative management of otitic brain lesions.

CEREBELLAR ABSCESS.

As we have seen from the statistics of various investigators (p. 358), cerebellar abscesses constitute about one-third of all otitic brain abscesses. Of these, not more than 15 or 20 per cent. are the result of acute tympano-mastoid disease, the remaining 80 or 85 per cent. being a complication of chronic middle-ear suppuration.

Symptoms.—As with most acute intracranial lesions, the symptoms may be considered under two heads,—viz., *general symptoms* of intracranial disturbance or compression; and *focal symptoms,—i.e.,* those which point definitely to the cerebellum as the site of the lesion.

The *general symptoms* are so nearly identical with those already described in connection with temporosphenoidal abscess that they may be reviewed very briefly. They include headache, vomiting, temperature and pulse changes, rapid emaciation, eye-ground changes, mental dulness, etc.

The *headache* in typical cases differs from that of temporosphenoidal abscess chiefly in that it is usually more severe, more persistent, and more frequently confined to the side of the head and to the regional area of the lesion. It may occur as an early symptom or only after the lesion is well advanced. Once it has made its appearance, it is usually an exceedingly persistent and harassing symptom. *Vomiting* may or may not be present. This fact, however, should be remembered,—viz., that recurrent vomiting (*i.e.,* coming at intervals throughout the entire course of the disease) is far more common with cerebellar abscess than with abscess of the temporosphenoidal lobe, in which vomiting is exceedingly rare except at the onset. In a case coming under the writer's observation, the two symptoms above mentioned—*i.e.,* persistent headache and recurrent vomiting—were during the greater portion of the attack practically the only symptoms pointing to endocranial disease. The *temperature and pulse changes* are much the same as in cerebral abscess. Fever is usually absent or inconspicuous, unless the meninges are also involved. A temperature range occasionally falling below the normal line is common with cerebellar as with cerebral abscess. Bradycardia is said to be a very frequent symptom of cerebellar abscess (Neumann); according to my observation and reading of the literature, it occurs in about one-third of all cases. *Changes in the eye-grounds*—papillitis, choked disk, optic neuritis—are much more frequently present in cerebellar than in cerebral abscess (Ruttin, Neumann); and the optic neuritis of cerebellar abscess is very frequently of such grade as to cause considerable, though not usually permanent, impairment of sight (Macewen). *Rapid emaciation* is a striking feature in many cases of cere-

bellar abscess. Occurring, as it frequently does, in cases in which fever is absent or of very moderate grade, it must be explained as a trophic change, due probably to compression or disturbance of some centre strongly influencing tissue metabolism. *Mental lethargy, dulness, or obscuration* is not usually present in cerebellar abscesses of moderate size, and when present probably always indicates an abscess of sufficient size to have induced marked increase in general endocranial pressure.

Muscular paralysis is a rare symptom of cerebellar abscess. Körner has pointed out that transmission of pressure from a cerebellar abscess to the cortical motor areas would be strongly resisted by the tentorium except in the case of very large abscesses. On the other hand, muscular paralysis or paresis might easily result from compression of the pons or medulla by a cerebellar abscess of comparatively small size. Such paralyses have been observed, sometimes contralateral and in other cases on the same side as the lesion. Macewen[34] explains these differences as corresponding to the level at which the motor tract is compressed. He points out that the cerebellum passes to some extent into the foramen magnum, and may extend further under abnormal pressure. On this hypothesis, he explains the occasional contralateral paralyses as due to pressure upon the pons, and the otherwise confusing paralyses on the side of the lesion as due to pressure upon the medulla at a level below the crossing of the motor tracts. Probably in this way must be explained the hemiparesis on the same side as the lesion which has occasionally been noted in cerebellar abscess. In the cases which have come within the personal knowledge of the writer moderate hemiparesis has been not uncommon. Genuine hemiplegia, on the other hand—*i.e.*, actual paralysis—has not been present.

Focal Symptoms.—It is necessary to state in advance of any discussion of the focal symptoms that in many cases they are absolutely wanting. In such a case the diagnosis may be exceedingly difficult. On the other hand, when focal symptoms are present, there is no intracranial lesion of which the clinical picture is more definitely characteristic. Among the symptoms entering into this symptom-complex are (1) nystagmus, (2) subjective vertigo, (3) incoördination ataxia on the side corresponding to the lesion, (4) diadokokinesis (Babinski), (5) loss or impairment of the arthrodial sense, or sense of position, (6) disturbance of static equilibrium. These phenomena call for brief comment.

The nystagmus of cerebellar abscess is of distinctly vestibular type,—*i.e.*, it is composed of a quick movement in one direction and a slow movement in the other. It is usually rotary in character. Superficially, therefore, its resemblance to the nystagmus of labyrinthine disease is complete. Its direction, however, is not regularly either toward or away from the side of the lesion. Very frequently it is a changing nystagmus, the direction of the quick component being first to one side and then to the other in accordance with the direction in which the eyes are voluntarily turned.

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[34] Macewen: Pyogenic Diseases of Brain, etc., p. 195.

Usually, however, the nystagmus toward the side of the lesion is stronger than the opposite nystagmus induced by rotating the eyes in the opposite direction. It is a progressive or increasing nystagmus, which usually shows no tendency to gradual subsidence, but tends rather to become more pronounced with the progress of the lesion (Neumann). While, therefore, the eye movements are identical in character with those seen in acute suppurative labyrinthitis, there are usually certain features which definitely differentiate the nystagmus of cerebellar disease from that induced by an irritative lesion of the vestibular apparatus.

Subjective vertigo is a more or less prominent symptom in most cases of cerebellar abscess. When nystagmus appears, there is probably at first a typical rotary vertigo, presumably induced by the nystagmus. According to Neumann, this subjective rotary vertigo tends to subside as the individual becomes inured to a nystagmus which is a stationary or increasing phenomenon of the disease. Even after this rotary nystagmic vertigo has disappeared, however, there usually remains a subjective disturbance of equilibrium which is quite independent of the nystagmus. It is also a fact that certain patients, who exhibit no nystagmus, yet suffer from more or less constant dizziness, or vertigo. I believe that this is an important symptom in certain cases in which the more characteristic focal signs are absent. That is to say, in a case of intracranial lesion without definite localizing symptoms, persistent vertigo would point, other conditions being equal, to the cerebellum as the probable site of the lesion.

Unilateral incoördination ataxia is usually present in cases in which any clear focal signs are demonstrable. It is shown by a lack of precision in certain movements of the hand and arm of the side corresponding to the brain lesion. It is best elicited by the common experiment of requiring the patient with eyes closed to touch the tip of the nose quickly first with the tip of the right forefinger and then with the left. It will be found that, while he can execute this movement with ease and precision with the finger corresponding to the sound side of the brain, the forefinger corresponding to the side of the lesion may miss the nose by some inches and is brought into contact with it only after a series of to-and-fro oscillations in its immediate vicinity.

Diadokokinesis (*Babinski*).—Regularly with incoördination ataxia will be associated the condition known as diadokokinesis. If we direct the patient to practise any common movement of the two hands, fingers, or wrists simultaneously,—*e.g.*, the finger movements as in piano practice,—it will be found that the one hand executes these movements quickly and with normal ease while the fingers of the hand corresponding to the cerebellar lesion execute the same movements in normal enough rotation, but slowly and with apparent labor. If we vary this experiment by asking him to rotate both wrists as rapidly as possible back and forth, the one wrist will be rotated rapidly while the other—corresponding to the side of the lesion—follows with similar rotations performed very slowly. In a case of right-sided cerebellar abscess coming under the writer's care, the

left wrist executed just four rotations while the right rotated once,—a ratio which for the average normal individual would be rather a difficult feat.

Loss or Impairment of Arthrodial Sense, or Sense of Position.—This may be tested by blindfolding the patient, passively moving the arm or leg corresponding to the lesion in different directions, and leaving it in some unusual position. He is then directed to duplicate its position with the corresponding opposite limb. Inability to do so shows loss of position or posture sense on the side corresponding to the lesion. It is a corroborative sign which, I believe, is often absent when other definite focal symptoms are present.

Disturbance of Equilibrium; Cerebellar Ataxia.—I have seen cases of cerebellar abscess in which the patient has been able to stand and walk steadily and without noticeable difficulty in maintaining his equilibrium. In other cases—and always when other focal symptoms are present—disturbance of static and dynamic equilibrium is a more or less prominent symptom.

In speaking of the ataxia of cerebellar abscess, Neumann states that "it stands in exact physiological relationship, demonstrable every moment, to the direction of the nystagmus and the position of the head in regard to the body. If, for example, rotary nystagmus toward the left side is present, the patient leans toward the right on standing erect," etc.[35] This conception of cerebellar ataxia is one which, in the opinion of the writer, will not be borne out by a careful study of any large series of cases.

With the first appearance of cerebellar nystagmus, subjective rotary vertigo resulting directly therefrom is probably always present, and this rotary vertigo may to some extent influence the patient's tendency to fall in a given direction. But it is admittedly a characteristic feature of cerebellar nystagmus that the related rotary vertigo tends to subside and finally to disappear. With disappearance of the nystagmic vertigo, the persisting ataxia can hardly be ascribed to the nystagmus, and as a matter of fact it may show no relation to the eye movements. I can illustrate this point by a case of cerebellar abscess of which I had the opportunity to make a very careful study.[36] This patient exhibited a nystagmus increasing with the progress of the lesion, and changing its direction in accordance with the direction in which the eyes were voluntarily turned, but very much more pronounced in the direction of the side of the lesion. So far as I could observe, and from the patient's statements, the nystagmus had little or no influence upon her subjective vertigo. When the patient attempted to stand she fell, or without support would have fallen, to the right,—*i.e.*, toward the side of the lesion. Throughout her illness her tendency to fall to the right was constant, which, it will be noted, was exactly opposite to

[35] Neumann: Cerebellar Abscess, Lake's translation, p. 23.
[36] Kerrison: Annals of Otol., March, 1911.

what should have been her direction of falling in relation to the nystagmus present. I believe that this is an important feature of the characteristic ataxia of cerebellar abscess,—viz., that, *when disturbance of equilibrium is a prominent and constant symptom, the direction in which the patient falls, or tends to fall, is constant and is for the most part independent of the nystagmus present.* Nor should this surprise one, for with incoördination ataxia, disturbed arthrodial sense, and in some cases hemiparesis, all confined to one side of the body, it seems not illogical to expect that these conditions should be not without influence in determining the direction in which the patient may fall. Logically, an ataxia which is not dependent upon an existing nystagmus, or in a case in which spontaneous nystagmus is not present, will not be influenced by changes in the position of the patient's head.

Naturally all symptoms may be modified or masked as the terminal stage is approached, when the patient's muscular reactions may be obscured to a point where, if placed upon his feet, he will fall in any direction according to the influence of gravity as determined by the chance position of his body.

Other symptoms of cerebellar abscess which, though inconstant, are characteristic and add when present to the completeness of the clinical picture, are *frequent involuntary yawning,* and *slow, thick articulation.*

An important group of focal symptoms which logically belongs here is that which includes the various disturbances of normal pointing accuracy,—sometimes properly spoken of as "past pointing." Their appreciation, however, is so clearly dependent upon a knowledge of Professor Barany's theory of cerebellar localization, that it seems better to devote a separate chapter to their consideration (Chapter XIV).

The Terminal Stage.—In its terminal stage a fatal case of cerebellar abscess may present much the same clinical phenomena as those already described in connection with temporosphenoidal abscess. The most characteristic difference is found in the respiratory changes, which are not very common in cerebral abscess but are particularly characteristic of cerebellar lesions. As death approaches the breathing may become slow, sighing, or even Cheyne-Stokes in character, or it may be simply stertorous. The end may come only with the final exhaustion of the patient's last vestige of vitality and resistance power, or may be hastened by rupture of the abscess into the subarachnoid space. In either case the immediate cause of death is usually respiratory paralysis.

Differential Diagnosis.—The two lesions for which otitic cerebellar abscess may be mistaken are *temporosphenoidal abscess* and *acute diffuse suppurative labyrinthitis.* When focal symptoms are absent it may very closely resemble a temporal-lobe abscess. With the appearance of focal symptoms the resemblance to cerebral abscess recedes, and the necessity for excluding suppurative labyrinthitis arises.

Differential Diagnosis between Temporal-lobe and Cerebellar Abscess.—Since either of these lesions may run its course with almost complete

absence of localizing symptoms, it may be necessary to weigh carefully even minor points which may throw light upon the site of the lesion.

Headache, for example, is common to both lesions. The headache of cerebellar abscess, however, is on the average more severe, less subject to periods of amelioration, and is more frequently confined to the occipital region on the side of the lesion. *Vomiting* characterizes the onset of both lesions. After the initial stage, vomiting is exceedingly rare as a result of temporal-lobe abscess, whereas it not infrequently recurs at indefinite intervals throughout the course of a cerebellar abscess. *Mental dulness* or *obscuration*, as shown by delayed or slow cerebration, failure of the power of sustained attention, etc., is a characteristic and in some cases a fairly early symptom of temporosphenoidal abscess. It is comparatively uncommon in cerebellar abscess, and rarely appears except as a late symptom and in company with other evidences of marked increase in general intracranial pressure. *Eye-ground changes*—papillitis, choked disk, or optic neuritis—are very much more common in cerebellar than in cerebral abscess, a fact which in certain cases may have some diagnostic significance. *Muscular paresis*, when present in temporosphenoidal abscess, is always contralateral. Hemiparesis on the same side of the body has frequently been noted in cerebellar abscess. *Vertigo* in temporal-lobe abscess is usually an inconstant and rather indefinite symptom, depending upon increase in general intracranial pressure. Noticeable disturbance of static equilibrium is not usually present in the early stages of the lesion. On the other hand, the vertigo and ataxia of cerebellar abscess may appear as prominent and fairly early symptoms. That is to say, the subjective disturbance is persistent, and the ataxia is usually characterized by a constant tendency to fall in a given direction.

When focal symptoms are present, the diagnosis becomes a comparatively easy matter. Thus, *sensory aphasia*—especially if occurring early in the course of a suspected intracranial lesion—points fairly definitely to a lesion of the left cerebral hemisphere, or, in the case of a left-handed individual, to the right brain. Aphasia does not occur in cerebellar abscess except as a late symptom and only in cases in which other signs of very marked general increase in intracranial pressure are present. Naturally, when focal symptoms of cerebellar abscess are well marked,—*e.g.*, cerebellar nystagmus and ataxia, incoördination ataxia, and disturbed arthrodial sense on the side corresponding to the lesion,—the case no longer presents difficulties of diagnosis.

The differential diagnosis between cerebellar abscess and diffuse suppurative labyrinthitis usually presents little or no difficulty to the careful student of labyrinthine and intracranial diseases. When focal symptoms of cerebellar abscess are absent, the clinical phenomena of this lesion in no way resemble those of suppurative labyrinthitis. Again, when the acute stage of suppurative labyrinthitis is passed, all clinical resemblance to cerebellar abscess ceases. When, however, focal symptoms of cerebellar abscess are present, this lesion and the acute stage of suppurative labyrinthitis may

present certain points in common,—*e.g.*, nystagmus, vertigo, static ataxia, vomiting, etc.,—which might lead to serious error. The chief differential points are briefly stated below.

Diffuse Suppurative Labyrinthitis (Acute Stage).	*Cerebellar Abscess.*
Rotary nystagmus toward sound ear; is always increased when eyes are voluntarily turned toward sound ear,—*i.e.*, in the direction of quick eye movement, being diminished or disappearing wholly when they are turned in the direction of the slow eye movement.	Changing rotary nystagmus,—*i.e.*, direction of quick eye movement may vary at different times; frequently changes in accordance with the direction in which the eyes are voluntarily turned. Usually, however, the most pronounced eye movements observed are those toward the side of the lesion.
The nystagmus tends regularly toward gradual subsidence, and usually disappears wholly in from 10 days to 2 or 3 weeks.	There is no tendency toward subsidence, the nystagmus usually becoming more marked with the progress of the lesion (Neumann).
Subjective rotary vertigo, always in the plane of the nystagmus, is increased when the eyes are turned in the direction of the quick eye movement; shows progressive diminution, and ceases wholly when the spontaneous nystagmus has completely disappeared.	Vertigo, at first influenced by the nystagmus, later ceases to be influenced thereby. Subjective vertigo is often present and persistent, even in cases in which nystagmus is not present.
Ataxia: there is a tendency to fall in the plane of the nystagmus and in the direction opposite to the quick nystagmic movement. This disturbance of equilibrium regularly subsides, and ceases wholly when the nystagmus has disappeared.	The ataxia, as a rule, shows no tendency to subside. Direction of falling is usually constant, and has no necessary relation to character or direction of nystagmus present.
Impairment of the touch-direction-sense; effort to point with either hand, eyes being closed, resulting in deviation in a direction opposite to that of the quick nystagmic movement. [30a]	When other focal symptoms are absent, touch-direction-sense, shown by accuracy in pointing with eyes closed, may be unimpaired. With focal symptoms, loss of accuracy in pointing—*i.e.*, deviation of hand to one side or the other—is usually present, but only in the hand or finger of same side as the cerebellar lesion. [30a]
Caloric reaction absent.	Caloric reaction usually normal.
Hearing absolutely lost in diseased ear.	Hearing normal, or at least not influenced by cerebellar disease.
Incoördination ataxia and diadokokinesis absent.	Incoördination ataxia and diadokokinesis may be prominent symptoms.
Arthrodial sense, or sense of joint position, not usually impaired.	Arthrodial sense may be lost, or markedly impaired.
Temperature, elevated at onset, shows in uncomplicated cases progressive and fairly rapid recession. Rarely subnormal.	Temperature, elevated at onset, usually recedes, and may then follow a fairly normal line, or may show moderate movement between points a little above and slightly below normal.
Pulse-rate may be normal, or varies in normal ratio with temperature changes.	Pulse-rate is not infrequently abnormally slow. Bradycardia may coexist with elevation of temperature.

[30a] For theory and significance of pointing, see next Chapter.

When the latent stage of diffuse suppurative labyrinthitis is reached the disease no longer bears any resemblance to cerebellar abscess. The differential diagnosis is then definitely established by the complete deafness and absence of caloric irritability in the diseased ear in suppurative labyrinthitis, hearing and vestibular irritability—*i.e.*, in response to heat and cold—being usually uninfluenced by cerebellar abscess.

OTITIC MENINGITIS.

Otitic meningitis is an elastic term which may very properly include a variety of conditions, from the circumscribed pachymeningitis, which commonly yields very promptly to rational treatment, to the diffuse, or generalized, purulent leptomeningitis, which ends in death.

Circumscribed Pachymeningitis.—This condition may be dealt with briefly. Every aural surgeon of considerable experience has during operation upon the mastoid met with occasional cases in which circumscribed areas of dura have been found thickened, congested, or even covered with granulations. The condition is not infrequently met with as part of the pathology of an extradural abscess, or it sometimes results from an erosion of the inner plate of the skull even when no distinct pus collection is found. Such areas of pachymeningitis may involve the dura over the tegmen antri (temporal lobe), the dural covering of the sigmoid sinus, or less frequently the dura covering the cerebellum. Frequently the condition is not suspected prior to operation. In the occasional cases in which severe pain leads to a tentative diagnosis of intracranial disease before operation, the pain is usually due to tension upon the dura by a considerable collection of pus rather than to the inflammatory process itself. The great majority of cases undergo prompt resolution as soon as all diseased bone is removed and the inflamed dural surface is freely uncovered. The subsequent treatment is simply the observance of surgical cleanliness, the wound being lightly packed at each change of dressing with sterile gauze until the involved dural surface is covered by a layer of uniformly healthy granulations.

So frequently is circumscribed pachymeningitis met with in cases in which no meningeal symptoms have been present, and so prompt is the usual response to treatment, that one might be led into the error of belittling the gravity of the lesion. It is a simple lesion only under prompt rational treatment. Without surgical intervention it is undoubtedly the precursor in many cases of the more serious lesions which jeopardize the patient's life.

Leptomeningitis resulting from suppurative middle-ear or mastoid disease, and producing symptoms sufficiently characteristic to be recognized as meningeal, was once regarded as an absolutely hopeless condition. During the past ten or fifteen years, however, the record of authenticated recoveries has reached a sufficient number to necessitate a modification of this view as to the hopelessness of the prognosis in all cases. In order to explain the varying response to treatment in cases often closely related

clinically, it is necessary to bear in mind certain variations in the character or extent of the morbid changes present, in accordance with which we may recognize the existence of different types or forms of the disease.

The three most important forms of otitic meningitis may be described under the following names: (1) Diffuse, or generalized, purulent leptomeningitis; (2) circumscribed purulent leptomeningitis; and (3) serous meningitis. While it is admittedly not always possible to determine definitely from its clinical aspects to what class a given case belongs, there are some cases in which this may be determined with reasonable certainty. Furthermore, the recognition of the existence of the three forms supplies a useful working hypothesis, and enables us to adopt rational methods of treatment resulting in a certain percentage of recoveries in cases which would otherwise terminate fatally.

Diffuse Purulent Leptomeningitis.—By diffuse purulent leptomeningitis we mean a suppurative inflammation of the meninges which has reached the subdural and subarachnoidal spaces, involving the arachnoid and pia mater, and which has spread widely—*i.e.*, with no tendency to limiting peripheral adhesions—within the meshes of the arachnoid.

The pia mater is very richly supplied with nerves and blood-vessels, its arteries separating into very numerous branches which pass directly into the brain cortex. A suppurative process involving the pia mater is thus carried directly into the outer layer of brain substance. Severe diffuse purulent leptomeningitis is, therefore, of necessity accompanied by a cortical encephalitis.

Paths of Infection.—The spread of infection from a suppurative focus in the middle ear or mastoid may occur by any of the following routes: (1) By direct infection through contact of necrotic bone with the dura, or through erosion of the inner plate at some point,—*e.g.*, of the tegmen tympani or the bony plate separating the mastoid cavity from the cerebellum. In such a case the meningeal lesion probably always begins as a circumscribed pachymeningitis. (2) Through an infected labyrinth, pus reaching the meninges (a) by way of the nerve channels opening into the internal auditory meatus; (b) by the aquæductus vestibuli, causing first an empyema of the saccus endolymphaticus; or (c) by way of the aquæductus cochleæ, which, it will be remembered, communicates directly with the subarachnoidal space. (3) A third pathway of infection is through an intermediate suppurative lesion of the brain. Every experienced surgeon will agree that a very considerable proportion of all fatal cases of brain abscess are toward the end complicated by purulent meningitis.

Symptoms.—The symptoms of otitic meningitis do not differ materially from those induced by meningeal inflammations arising from other causes. They may, therefore, be dealt with briefly. The onset is usually announced by a chill, or rigor, a sharp rise of temperature, and in many cases by vomiting, with or without nausea. Headache, always an early symptom, is usually severe and may persist throughout the course of the disease. The pulse is usually accelerated, in adults reaching 120 to 140,

while in children it may be even more rapid. The face is often noticeably flushed. The patient is usually exceedingly restless, and frequently exhibits an irritability of temper which may be in striking contrast to his normal mental state. These symptoms, then,—viz., recurrent vomiting, severe headache, high fever, rapid pulse, and restlessness,—commonly characterize not only the onset, but also the first two or three days of the attack. The clinical picture even to the superficial observer is one of severe illness, and in a case of tympanic or mastoid disease clearly indicates a serious change for the worse.

Usually by the second or third day we have the following additional symptoms: There is noticeable rigidity of the muscles of the back of the neck. This soon increases to the point where, if one tries to flex the head forward so that the chin will rest upon the sternum, this is resisted, and we may to some extent lift the shoulders before the neck muscles will yield. Kernig's symptom is often, though not always, present,—*i.e.*, if the knee is bent and the thigh flexed toward the abdomen, passive straightening of the leg is rigidly resisted. Photophobia becomes sooner or later a noticeable symptom. The mental condition, from one of extreme "restlessness" or "irritability," soon lapses into delirium. Delirium, at least during the night, is usually a fairly early symptom in diffuse meningitis. From this stage, the course of the disease is usually an exceedingly rapid one and provides one of the most distressing and discouraging pictures of fast ebbing of the life current that the physician is called upon to witness. The patient tosses distressfully about the bed, happily without knowledge of his condition and often, it is to be hoped, without conscious suffering,—the extreme restlessness subsiding only as he gradually sinks into coma, the immediate precursor of death.

Pupillary changes are very common, the most frequent being contraction of the pupil corresponding to the side of the lesion. Occasionally there is strabismus, usually inward. The pupillary changes, while in some instances affecting both eyes to some extent, in the majority of cases are unilateral or more marked in the eye on the side of the lesions.

Changes in the eye-grounds—*e.g.*, choked disk, papillitis, optic neuritis —are very often present in diffuse leptomeningitis.

The above group of symptoms, while fairly typical of the average course of the disease, is naturally subject to many clinical variations. It must be remembered that the onset, as we recognize it, may not represent the incipiency of the lesion, but simply its first clinical manifestation. Rigidity of the neck, photophobia, and even delirium may, therefore, in some degree be noticeable almost from the start. The pulse, which is usually very rapid, is in exceptional cases abnormally slow,—*e.g.*, 64 or 60 beats per minute or less,—this phenomenon being presumably due to a coincident cortical encephalitis with possible disturbance of some centre inhibiting cardiac activity.

One of the most characteristic features of the clinical picture of diffuse purulent meningitis is the sudden and striking change which occurs in the

patient's appearance at a certain stage of the disease. To-day he looks out upon the world through intelligent eyes, showing anxiety about his condition and perhaps a realization of its gravity. To-morrow he is delirious and has stamped upon his face the unmistakable sign of impending death.

Blood Count.—A characteristic feature of purulent meningitis is a high and increasing leucocytosis with a high percentage of polymorphonuclear cells.

Lumbar Puncture.—In the first stages of a meningeal attack, lumbar puncture may furnish the only reliable data as to the nature and gravity of the lesion. That is to say, the presence of pus and bacteria in the spinal fluid may point definitely to purulent meningitis at a stage when the clinical picture is yet incomplete.

Prognosis.—When a suppurative leptomeningitis has become diffuse,—*i.e.*, has spread widely in the meshes of the arachnoid,—death is inevitable.

Circumscribed Leptomeningitis.—This term is used to describe a suppurative inflammation involving the arachnoid and pia mater over a distinctly limited area, lateral extension being prevented by marginal adhesions binding the arachnoid and pia together and to the outer dural layer; or it may be that marginal extension is in some cases prevented by the combination of a mild local infection with high powers of local resistance. The marginal limitation of the lesion, while preventing the development of widespread, or diffuse, leptomeningitis, does not necessarily insure that some of the germs responsible for the infection shall not find their way into the fluid of the spinal canal. Lumbar puncture may, therefore, yield fluid containing pus and bacteria, *and the presence of pus and bacteria in the cerebrospinal fluid may constitute no proof of the existence of what may properly be called diffuse purulent leptomeningitis.*

Unfortunately, the symptoms of circumscribed leptomeningitis may follow practically the same course and sequence as those described as characteristic of diffuse leptomeningitis. Assuming that the lesion remains circumscribed, we might expect the progress of the disease to be less rapid, and this is undoubtedly a differential clinical point in certain cases. Unfortunately, even were we able to determine positively in a given case the circumscribed character of the disease, there would be no way of determining that adhesions might not at any moment be broken down and the disease thus quickly converted into a widely spreading, and therefore rapidly fatal, form of diffuse leptomeningitis.

There can be no doubt that many cases of circumscribed leptomeningitis of otitic origin, which would otherwise have ended fatally, have been cured by timely operative intervention. This proposition is no longer an hypothesis, many authenticated recoveries recorded during the past ten or fifteen years supplying a basis of actual knowledge. Jansen,[37] as far back as 1895, recorded a recovery in a case in which the lesion was clearly proved to be

[37] Jansen: Berlin. klin. Woch., 1895, No. 35, pp. 763–765.

circumscribed leptomeningitis. This patient, having suffered for years from chronic suppurative otitis media of the left ear, suddenly had a chill, high fever, and severe headache. Coincidently he developed symptoms of sensory aphasia which naturally led to a tentative diagnosis of abscess of the left temporal lobe. Following quickly upon the symptoms mentioned, the patient became delirious. An exploratory operation for temporosphenoidal abscess was then undertaken. No abscess was found in the brain, but the dura and pia were discolored, adherent, and a drop of pus escaped from beneath the dura when the membranes were incised. Recovery proved the condition to have been one of circumscribed leptomeningitis.

Lucae[38] in 1899 reported a case in which the diagnosis of otitic meningitis was based upon the symptoms. At the operation the exposed dura presented an area of greenish discoloration, from the meshes of which, when incised, pus exuded. The patient made a slow but perfect recovery.

Gradenigo[39] has reported two cases presenting typical symptoms of leptomeningitis, in which the fluid from the spinal canal contained staphylococci, which recovered after operation.

Similar cases have been recorded in this country by Arnold Knapp,[40] McKernon,[41] Kopetsky-Held,[42] and others. These cases, cited at random from the literature, are sufficient to establish this important fact,—viz., that there are certain cases of purulent leptomeningitis of otitic origin which are curable by surgical means. This naturally introduces the question as to what type of purulent leptomeningitis is curable and in what class is the prognosis hopeless. Some surgeons—among whom are Lemoyes and Gradenigo—have regarded the cases recovering after operation as cases of diffuse leptomeningitis, and have naturally, therefore, held a more optimistic view as to the prognosis in the diffuse form of the disease. On the other hand, Heine cites Hinesberg, Bertelsmann, Buschmann, and Grossmann (most of whom have operated successfully in cases of otitic meningitis) as sharing with him the opinion that all these cases of recovery have belonged to the circumscribed form of purulent meningitis, and that diffuse suppurative leptomeningitis is a hopelessly fatal disease which the surgeon is powerless to relieve. To the writer this view seems to be the only one tenable. Several cases of recovery from otitic meningitis have come within his personal knowledge, but none in which a positive diagnosis of diffuse suppurative leptomeningitis could be made. We must remember that in the diffuse form of the lesion the infection travels marginally through the communicating spaces, or meshes, of the arachnoid (Hinsberg). When we recall the postmortem findings as we have all seen them in some case of rapidly fatal diffuse meningitis,—the arachnoid adherent

[38] Lucae: Berlin. klin. Woch., 1899, No. 23, pp. 497–499.
[39] Gradenigo: Arch. für Ohrenheil., Bd. 47, p. 155.
[40] Knapp, A.: Arch. of Otology, vol. xxxv, No. 1.
[41] McKernon: Arch. of Otology, vol. xxxvii, No. 3 and 4, 1908, pp. 183–189.
[42] Kopetsky-Held: Arch. of Otology, vol. xxxv, No. 6.

over a wide area both to the dura and the pia mater, and its meshes everywhere infiltrated with thick pus,—the hopelessness of surgical treatment seems clear.

Serous Meningitis; Serous Meningo-encephalitis (**Körner**).—Still another form of meningeal lesion of otitic origin is that known as serous meningitis. It may be secondary to a circumscribed pachymeningitis such as frequently occurs with extradural abscess. Quite frequently, however, no such intermediate lesion is in evidence. Serous meningitis is present in those cases of meningeal inflammation in which the arachnoid and pia mater, though congested and œdematous, have escaped actual infection. This secondary œdema may involve the adjacent brain surface (serous encephalitis), and may cause very considerable increase in intracranial pressure. The symptoms may follow very closely those of diffuse purulent leptomeningitis. A differential point between serous and purulent meningitis is the fact that pus and bacteria are not found in the cerebrospinal fluid in purely serous meningitis. On the other hand, bacteria may also be absent from the spinal fluid in cases of purulent meningitis. Lumbar puncture, while supplying very important data, does not, therefore, always enable us to arrive at a positive diagnosis. In a case coming under the writer's care, the clinical picture was quite typical of purulent meningitis, and this diagnosis was made by competent surgeons in consultation. That it was really a case of serous meningitis was determined only after the patient's recovery was assured. Indeed, one of the most important points in the diagnosis of serous meningitis is the recovery of the patient.

The symptoms vary somewhat in accordance with the depth to which the subdural tissues are involved. Thus, when the congestion and œdema involve only the arachnoid and pia mater, the symptoms are meningeal in character, this constituting serous meningitis in its most typical form. In such a case the clinical phenomena may be practically the same in kind as those usually characterizing purulent leptomeningitis. If, however, the œdema extends well into the subjacent cerebral cortex, a train of symptoms more characteristic of cerebral disease—*e.g.*, somnolence, retarded cerebration, or possibly aphasic phenomena—may preponderate. To such a lesion the term *serous encephalitis* has been applied.

Perhaps a brief outline of a few typical cases will serve better to convey a correct idea of the lesion than any amount of formal description.

Case I.—In June, 1904, the writer was called to see a girl of five years, who was suffering from acute suppurative mastoiditis of the right side. The lesion had reached a stage (postauricular abscess) requiring immediate operation. Removal of the cortex revealed pus and granulations in the antrum and many of the mastoid cells. Three days later, severe headache was complained of, the temperature rose to 104° F., the neck became somewhat rigid, and the child became delirious. On the following day there was internal strabismus. Dr. A. B. Duel in consultation concurred in the diagnosis of otitic meningitis. An immediate operation was performed, which consisted merely of careful exposure of the dura covering

the temporal lobe by removal of the tegmen tympani et antri and contiguous portions of the squama. The dura showed no structural changes macroscopically, and it was decided to await further developments before incising it. Following this operation, the meningeal symptoms began to subside, and the patient made a perfect recovery. The symptoms in this case can be explained only as having been caused by a serous meningitis.

Case II was that of a young woman, twenty-three years of age, who came under my care as a ward patient in the Manhattan Eye, Ear, and Throat Hospital in 1907. A radical operation for chronic suppuration of the right ear had been performed one year previously in a neighboring city. At the time of her admission to the Manhattan Hospital, her condition was about as follows: There was a slight discharge from the right ear. There were no focal symptoms pointing to any particular intracranial lesion. Mental condition decidedly dull, cerebration somewhat obscured, but she answered all questions intelligently. She was disinclined to any muscular exertion, and when required to stand or walk was noticeably unsteady. The patient's lack of interest in her surroundings and in her own condition extended to the matter of food: she was without appetite and it was difficult to induce her to take sufficient nourishment. Loss of flesh and strength was therefore rapid. Temperature ranged between 100° and 101° F.

The above symptoms, though indefinite, seemed to me to suggest abscess of the right temporal lobe rather than any other lesion, and I determined upon an exploratory operation. The dura covering the temporosphenoidal lobe was freely exposed by the removal of a considerable portion of the squama. No macroscopic changes in the dura were noted. A narrow-bladed knife was inserted into the temporal lobe in three different directions, but no abscess was found, and the operation was ended here. To the surprise of the writer, the patient from the time of this operation showed progressive improvement, and was discharged, cured, within a month. The clinical phenomena in this case can be explained upon no other hypothesis than that they were due to a serous encephalitis.

Case III[43] has been reported in full elsewhere, and will be given here only in brief outline. Mr. C., thirty-four years of age, had suffered from early childhood with chronic suppurative otitis media of the left side. On June 4, 1907, he had experienced severe pain in the left ear. On the following morning, when I first saw him, the temperature had reached 103° F., pulse 120. He had vomited at frequent intervals. Mental condition dull, but he answered correctly all direct questions. By the afternoon of the same day, he had become delirious, and did not recognize even members of his own family. There was some rigidity of the muscles of the neck. The pupils were contracted to pin-points. Two other physicians, called

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[43] Kerrison: Arch. of Otology, vol. xxxvii, No. 2, 1908.

in consultation, confirmed the diagnosis of otitic meningitis and advised immediate operation, which was performed as soon as the patient could be prepared.

Operation.—A radical operation was quickly performed, revealing a sclerotic bone and an antrum filled with thick, offensive pus. The roof of the antrum and portions of the squama were then removed, exposing an area of dura covering the temporosphenoidal lobe measuring from before backward about three inches. No pus was found between dura and bone. Increase in cerebral pressure shown by bulging of exposed dura. Dura somewhat engorged,—*i.e.*, showed small areas of capillary injection.

The dura was now opened by three parallel, vertical incisions about one inch apart. Considerable oozing of cerebral fluid followed. Through each of these incisions a narrow knife was successively introduced into the brain substance, that through the posterior incision reaching the lateral ventricle and giving exit to a considerable flow of cerebral fluid. This completed the operation. Loose sterile gauze was placed in contact with the incised dura, this being covered by a large sterile dressing.

On the following morning, the patient's temperature was 98.6° F., pulse 78. He complained of severe headache, but his mental condition was quite clear. He retained no memory of the happenings of the preceding day, which has remained a blank in his experience. For some weeks following the operation, there was a rather copious loss of cerebral fluid which ceased only when the dural openings finally closed. The patient made a complete recovery. In this case we have a perfect example of a severe type of serous meningitis which would undoubtedly have ended fatally but for the prompt and rather drastic surgical treatment.

Meningismus.—Before leaving the discussion of meningeal disorders which may have an otitic origin, passing mention should be made of a condition in which, without actual invasion by infective organisms and without grave pathologic change, the meninges are subjected to certain transitory irritative influences of sufficient violence to induce symptoms almost or practically identical with those characteristic of a severe meningeal inflammation. To this condition, the pathology of which is not clearly known, the name meningismus is applied. It may be induced, particularly in young children, by a purulent otitis media or mastoiditis, but is probably much oftener due to other causes.

Differential Diagnosis.—Having described briefly the three main types of otitic meningitis, one is forced to admit that it is rarely possible at the beginning of a meningeal attack to determine to what group it will prove ultimately to belong. It is probable that it will never be possible to make an early positive differential diagnosis between a circumscribed and a diffuse purulent leptomeningitis. Yet the importance of endeavoring to arrive at a correct diagnosis as early as possible will hardly be questioned. Everyone knows the inherent danger in the practice of any special branch of medicine,—*i.e.*, of viewing all diseases from the cramped and narrow confines of that specialty. While by no means an invariably

reliable index, it is beyond question that the character of the spinal fluid will supply in many cases of meningeal disorder the most definite diagnostic data at our disposal. To give such data their broadest value and usefulness, it is clear that the physician should have in mind not only the recognized types of otitic meningitis, but should consider as well—since intercurrent and quite unrelated lesions are always a possibility—whatever other conditions might give rise either to the clinical symptoms present or to changes in the character of the spinal fluid. It may serve a useful purpose, therefore, to state briefly (1) the character of the normal cerebrospinal fluid; and (2) some of the commoner disorders or lesions in which its character may undergo change.

The Cerebro-spinal Fluid in Health and Disease.—The conditions which an examination of the spinal fluid may help to differentiate are (a) epidemic cerebro-spinal, or meningococcic, meningitis; (b) purulent meningitis, circumscribed or local, *i.e.*, due to infection by other micro-organisms; (c) tuberculous meningitis; (d) meningismus; and (e) in times of epidemic, and probably always in the larger cities, anterior poliomyelitis.

A normal fluid is perfectly clear. During lumbar puncture, it flows slowly,—*i.e.*, about 10 to 15 drops to the minute.

In practically all diseased conditions involving the meninges, the spinal fluid is increased in amount.

A perfectly clear spinal fluid under pressure,—*i.e.*, showing considerable increase in amount—may mean meningismus, tuberculous meningitis or anterior poliomyelitis. It may also indicate syphilis of the spinal cord. As occasional exceptions to the above, slight cloudiness of the fluid is seen in certain cases of poliomyelitis and tuberculous meningitis.

Markedly cloudy fluids usually indicate meningitis due to the meningococcus (epidemic cerebro-spinal m.) or to any of the pyogenic micro-organisms (purulent m.). It is conceivable, however, that an inflammatory process in tympanum or mastoid might spread by continuity of tissue to the meninges without actual invasion by the pyogenic organism present. Such a lesion might give a cloudy fluid (serous meningitis).

In meningismus the cell count is not usually increased,—*i.e.*, not more than one cell to four or five fields is usually found.

In purulent meningitis, meningococcic meningitis and tuberculous meningitis, the cells are increased. The following differences, however, are to be noted, viz., (1) the cell increase is as a rule much greater in purulent or in epidemic cerebro-spinal meningitis than in tuberculous meningitis; and (2) in purulent and in meningococcic meningitis the polymorphonuclear cells preponderate by nearly 100 per cent., whereas in tuberculous meningitis, the mononuclear cells preponderate in nearly equal ratio (95 per cent.).

There is an increase in the albumen and globulin in the spinal fluid from cases of tuberculous, meningococcic and purulent meningitis. There is usually no increase in meningismus.

The presence of glucose, as shown by Fehling's test, varies from the

normal in certain diseases. There is usually a normal reduction by Fehling's test in poliomyelitis and meningismus; it is diminished in a majority of cases of tuberculous meningitis. In meningococcic and purulent meningitis it may be normal at the onset and in very mild cases ending in recovery may remain normal throughout the disease. In cases of average severity, however, the Fehling's reduction is regularly diminished or absent as the disease reaches its height,—increasing in cases ending in recovery as convalescence is established.

I wish to acknowledge my indebtedness for the above chiefly to Doctors DuBois and Neal. Their data, based on the results of seven years of work in the New York health board and on a correllation of the clinical and laboratory findings in 1600 cases, are of a kind which should carry unusual weight. Among practical deductions drawn from this experience, may be mentioned the following.

(1) Disappearance of sugar from the spinal fluid as shown by diminution or absence of the normal Fehling's reduction is not a reliable early test of purulent meningitis, since in the majority of cases it is not present until the disease is well advanced. Since a return of the normal reduction often marks a turning point in the lesion for the better, repeated tests may furnish useful prognostic data.

(2) In purulent and in meningococcic meningitis a high polymorphonuclear percentage is a characteristic feature of the spinal fluid. A gradual and progressive increase in the relative number of the mononuclear cells is a frequent concomitant of a change for the better and is a good prognostic sign.

(3) Progressive diminution of the albumen and globulin to a normal basis often keeps pace with a favorable change in the progress of the lesion.

(4) Favorable changes in the chemical and physical properties of the spinal fluid—*e.g.*, reduction of cell count, albumen and globulin, increase in sugar, etc.—without coincident disappearance of the pathogenic micro-organism, are unfavorable signs. Such cases usually end fatally.

With regard to the isolation by smears or cultures of the causative micro-organism in the spinal fluid, a positive finding of course definitely classifies the lesion. A negative finding in fluid from the first or second puncture does not, however, necessarily indicate a non-purulent meningeal lesion since the germ may be isolated from a specimen obtained from a later puncture.

Prognosis.—In speaking of the prognosis, one must necessarily take somewhat into account the various types of the disease, since in cases which recover the diagnosis is so often open to discussion. That purulent leptomeningitis is not a hopelessly fatal disease may be considered proved by the large number of authentic and trustworthy case reports in which recovery has taken place. It is conceivable that a meningeal infection involving only a small area may produce all the characteristic clinical phenomena and release pyogenic micro-organisms into the spinal fluid; and it is probable such circumscribed lesions have furnished a large proportion of the recoveries. Of the various micro-organisms which may reach the meninges from

the ear, the streptococcus mucosus capsulatus (now regarded as identical with the pneumococcus no. 3) seems to be the most fatal, few recoveries having been reported, so far as the writer knows, in which this germ was found in the spinal fluid.

Treatment.—It must be confessed that the surgical treatment of meningitis has not yet been placed upon a scientific basis. And yet, enough cases have been successfully operated upon to place upon the surgeon a grave responsibility in the treatment of cases. So far as a logical course of procedure may be formulated, it may be stated briefly as follows:

If an otitic origin is in doubt, a lumbar puncture might give important data. There is little question that cases of meningitis of the epidemic, or meningococcic, type coexisting with aural suppuration, have in some cases been operated upon on the hypothesis of an otitic origin. A positive finding of the meningococcus in the spinal fluid would supply definite indications as to treatment: *i.e.*, intraspinal administration of the antimeningitis serum, and no surgical intervention unless separately indicated for the mastoid lesion.

In cases in which the spinal fluid is increased in quantity but clear and otherwise approximately normal, the question of a transitory meningismus must be taken into account. Operative treatment may be absolutely contraindicated. On the other hand, it must be borne in mind that not only pneumonia, intestinal infections and the acute infectious diseases, but also localized foci of infection, may cause the meningeal disturbance. Elimination of a suppurative process in the mastoid may, therefore, be called for.

In cases of purulent leptomeningitis clearly traceable to an otitic origin, surgical treatment, to have any value, must not be delayed. Thorough removal of all diseased structures contiguous to the meninges is the first desideratum. This does not mean that time should be unduly expended upon the curetting of small sections of bone, but rather that diseased bone in contact with the dura should be freely removed in bulk, it being better to err in the direction of removing too much than of leaving necrotic or devitalized bone in contact with the already hyperæmic or inflamed dura. This preliminary work upon the bone should be done with all reasonable rapidity, speed being an important factor in operations involving the brain or meninges.

In the treatment of meningitis, serous or purulent, the older view as to the necessity in all cases of incising the dura requires modification. We now know that in many cases of serous, and in some cases of purulent, meningitis thorough removal of diseased bone and free exposure of the adjacent dura are all that are required to bring about recovery. Usually the roof of the antrum and tympanic vault and contiguous portions of the squama are the parts to be removed. Generally speaking, the dura should be uncovered well beyond the boundaries of the area supposed to be involved. Cases of recovery following this procedure have been recorded by Brieger, Hinsberg, Gragenigo and others in Europe and have been observed by many surgeons, including the author, in this country.

Should symptoms of serous encephalitis be pronounced, or in any case should the operation above described not result in fairly prompt relief of the meningeal symptoms, there should be no delay in incising the dura.

The macroscopic appearance of the exposed dura is not a reliable guide as to the presence or absence of a serous or purulent meningitis. Superficial congestion of the dura or even granulations thereon are often noted in cases in which all meningeal symptoms are absent, and, on the other hand, a dura of normal appearance may coexist with subdural œdema or actual infection.

Where incision of the dura is indicated, the best results are probably obtained by two or more short incisions extending into the subarachnoid space or into the cerebral cortex. No attempt to introduce a wick or drain of any kind beneath the dura or into the brain substance should be made, simple incision providing the relief of tension and withdrawal of excess cerebral fluid which the operation aims to secure.

While diffuse purulent leptomeningitis is probably a hopelessly fatal disease, it is not always possible to determine the diffuse character of the lesion from the clinical manifestations. I believe, therefore, that it is justifiable in every case to give the patient the advantage of operative intervention. Careful removal of all diseased bone and free exposure and incision of the involved area of dura constitute theoretically the best surgical treatment, and in certain cases not clinically distinguishable from diffuse leptomeningitis may bring about recovery.

In the earlier years of his experience the writer saw several cases of otitic leptomeningitis treated by incision and attempted drainage by the introduction of small wicks of folded gauze or folded rubber tissue beneath the dura, but never one that did not end fatally.

Lumbar Puncture for Relief of Pressure.—Lumbar puncture repeated at intervals for the purpose of relieving intracranial pressure has been more or less extensively advocated and practised in otitic meningitis. While certain cases in which it was used have recovered, there is no evidence that such favorable results have been to any extent attributable to the withdrawal of fluid from the spinal canal.

Use of Drugs.—As to the internal administration of drugs,—*i.e.*, either hypodermically or by mouth,—there is not the slightest evidence that they exert any influence whatsoever on the course of the disease.

CHAPTER XIV.

BARANY'S THEORY OF CEREBELLAR CENTRES: NORMAL ACCURACY IN POINTING WITH EYES CLOSED: NORMAL REACTION MOVEMENTS IN RESPONSE TO VESTIBULAR IRRITATION: LOSS OF POINTING ACCURACY AND CHANGES IN THE REACTIONS TO VESTIBULAR IRRITATION IN CEREBELLAR DISEASE.

No discussion of the symptoms of cerebellar abscess would be complete without reference to the important work of Professor Robert Barany, now of the University of Upsala, in establishing the theory of cerebellar localization. According to this theory, there exist in the cerebellar cortex, certain centres which exert each upon some particular joint or its controlling muscle group, a pull, or tonus, in a certain direction. The influence of these centres does not, as in the case of the cerebral motor centres, control the voluntary movements of the muscle group or groups over which they preside. Rather they have to do with orientation, or the sense of position of different parts of the body with reference to certain fixed points in space.

Each cerebellar centre exerts upon some particular joint or its controlling muscle groups, a pull, or tonus, in a certain definite and constant direction. For each joint there exists separate centres which maintain tonuses in different directions. There are, for example, separate centres which exert respectively an inward and an outward tonus, or pull, upon the muscle groups of the shoulder. The operation of these two tonuses, acting in normal balance or equilibrium, confers upon the individual the ability to move the arm in the vertical plane without the aid of sight. Logically in such a scheme there must be additional centres for each joint exerting respectively an upward and downward pull, to maintain correct orientation or movement in the horizontal plane.

Apparently the cerebellar cortex contains four rather large areas, or centres, which may be thought of, not with reference to the joints they control, but in accordance with the direction of the tonus, or pull, which they exert upon all the principal joints of the body. In other words, the separate centres exerting, for example, an inward tonus upon the various joints are contiguous, or at least in rather close proximity to each other, the area in which they are grouped being spoken of as a direction centre. Thus, there are direction centres for inward, outward, upward and downward pull, or tonus, respectively. Within each direction centre are smaller centres specialized for the different joints, *e.g.*, shoulder, elbow, wrist, hip, knee, ankle, the neck, etc.

As to the influence of disease upon one or any of these cortical cerebellar centres: let us take for an example the shoulder joint, in which the ability with eyes closed to move the arm correctly in the vertical plane depends upon

the proper and coördinated functioning of the two centres exerting respectively an inward and outward pull upon that joint. If, now, the centre for inward tonus is destroyed by surgical accident or disease, the arm no longer moves correctly in the vertical plane, but veers outward, being drawn thither by the unopposed force of the still intact centre for outward tonus.

In order to convert this theory of cerebellar localization into a practical working hypothesis, it will be necessary to pause here to state briefly certain facts and describe certain phenomena upon which the so-called pointing tests are based.

Normal Pointing Accuracy.—The average normal individual with eyes closed and having located some fixed object by the sense of touch—say with the index finger of either hand—can raise the hand and arm to the vertical or upright position and then bring the finger again into contact with the object touched, or will miss it only by a fraction of an inch. If, instead of raising the hand, the arm be moved in the horizontal plane so that the hand travels through one fourth of the arc of a circle, it can be brought back, again traversing the horizontal plane, to contact with the object touched. This represents the normal standard of accuracy.

Method of Applying Pointing Test.—The pointing test most commonly employed is of the accuracy of movement in the vertical plane and is applied as follows: the examiner and patient stand or sit facing each other, the latter with eyes blind-folded or closed and with the arm to be tested extended straight in front of him. The hand is pronated so that the palmar surface which is to receive the touch impression is directed downward. In contact with the palmar surface of the patient's forefinger, thus extended, the examiner places his own finger, pressing upward. The patient is now instructed to raise the hand and arm to the vertical (upright) position and then rather slowly lower his finger to contact with that of the examiner, which has been held immovably in the original position. The average normal individual finds no difficulty in doing this, or misses only by a fraction of an inch.

Normal Pointing Reactions in Response to Vestibular Irritation.—We are now in a position to study the character of the deviation from normal pointing accuracy which regularly accompanies sudden disturbance of either vestibular apparatus, either as a result of disease or of experimental stimulation.

Method of Applying Test.—If we irrigate with cold water, let us say, the right ear (patient's head being held erect), there is developed a well-marked rotary nystagmus to the left, and the individual, attempting to stand, tends to fall to the right. Now quickly, *i.e.*, while the nystagmus is still active, test his pointing accuracy in the vertical plane, first with one hand and then with the other, by the method above described. It will be found that, having located the examiner's finger with his own, the hand in being raised will swerve somewhat from the vertical plane to the right, and again in being lowered will deviate further to the right. His hand, then, in being elevated and depressed describes an inverted **V** falling to the right of the object he is trying to reach. Stating this in the form of a rule, we may say that *the point-*

ing deviation resulting from vestibular irritation is invariably in the direction opposite to that of the induced nystagmus. It therefore corresponds with the direction of the falling tendency,—*i.e.*, with the direction in which, if he attempted to stand, he would demonstrate a tendency to fall. In a normal person these reactions are present in both arms.

Changes in Pointing Accuracy in Cerebellar Disease.—Like other focal symptoms of cerebellar disease, changes in pointing accuracy may or may not be present. When present, they may be the deciding factor in an otherwise obscure case. Their absence, however, is no indication that the cerebellum is not diseased.

Application of Test.—The patient, being blind-folded or with eyes closed, the pointing accuracy of the two arms is separately tested by the method already described. In a case of cerebellar abscess in which the centre for inward tonus upon the shoulder is involved, it will be found that the arm corresponding to the cerebellar lesion will deviate outward while the opposite (*i.e.*, that corresponding to the sound side) will continue to point with normal accuracy. Thus in a lesion of the right cerebellar hemisphere, the right hand deviates outward, *i.e.*, to the right; with a left cerebellar lesion, the left hand deviates to the left, the arm corresponding to the sound cerebellar hemisphere maintaining in either case the normal pointing accuracy. This is the first part of the test. We must now confirm the result thus obtained by testing the reaction to vestibular irritation.

Supposing, for example, that in a case of suspected cerebellar abscess we have tested the pointing accuracy of both arms and found that the right shows unmistakable outward deviation (*i.e.*, to the right) while the left arm points accurately,—this, so far as it goes, would indicate disease of the right cerebellum. We must now test the reaction of the right arm to vestibular irritation. This is done by irrigating the left ear (*i.e.*, ear of opposite side to the supposed lesion) with cold water. This is followed by a rotary nystagmus to the right, during which in a normal individual both hands in pointing would deviate strongly to the left. If now in the presence of an induced nystagmus to the right, the right arm does not deviate as normally to the left, but continues as formerly to deviate to the right, while the left arm shows the normal reaction to the left, we have a clear and positive indication of a lesion involving the right hemisphere of the cerebellum.

In order to illustrate these reactions, it may be well, though many typical cases have since been reported, to cite only two or three of the classical cases given by Professor Barany in his original reports.

Case I.—This patient, a boy, exhibited some of the general symptoms suggesting an intracranial suppurative lesion, but (excluding pointing tests) no focal or localizing symptoms pointing to cerebrum or cerebellum or indicating in which side of the skull the lesion, if present, should be looked for. The pointing tests demonstrated the following conditions: The left arm maintained its normal pointing accuracy, and in response to vestibular irritation showed normal reactions. The right arm, on the other hand, exhibited constant spontaneous outward deviation, *i.e.*, to the right. When

the left ear was irrigated with cold water, the usual rotary nystagmus to the right was induced, but the right hand did not show the normal deviation to the left.

On these two functional changes,—viz., spontaneous outward deviation of the right arm and its failure to respond as normally to vestibular irritation, a diagnosis of right cerebellar abscess was made. This diagnosis was confirmed by an operation during which the abscess was located and evacuated.

Let us now note in this case the further functional changes which followed the opening and evacuation of the abscess. Two days after the operation it was found that the pointing accuracy of both arms was normal,—or, in other words, the spontaneous outward pointing deviation of the right arm had entirely disappeared. When, however, the reaction to vestibular irritation was tested, it was found that when the left ear was irrigated with cold water, the left arm showed the normal deviation to the left, while the right arm continued to point with normal accuracy. In other words, as a result of the cerebellar abscess or quite possibly of the incision through which it was evacuated, the right arm could no longer be made to deviate to the left in response to vestibular irritation.

Since Cases II and III (also from Barany's original reports) are cited chiefly for their bearing upon the position of certain centres, a brief digression to define their location may serve a useful purpose. As the cases from which our knowledge has been chiefly derived are for the most part those of cerebellar abscess of otitic origin, it is clear that the cerebellar surface extending from the anterior border of the sigmoid sinus forward along the posterior aspect of the petrous bone to the internal auditory meatus represents the general area which should first be investigated; and it is a fact that in the cases in which these phenomena have been most clearly demonstrable, the lesion has usually been found in close proximity to this area.

Cerebellar Centres for Wrist and Shoulder (Fig. 196).—Barany places the centre exerting an inward tonus, or pull, upon the wrist joint in the anterior end of the middle inferior lobe (slender lobe)—that portion of it which is nearest the flocculus (1). The centre exerting a similar tonus upon the shoulder joint is located also in the middle inferior lobe, but behind the wrist centre by about 10 or 12 mm. (2). The evidence as to the correct localization of these two centres will be stated presently. The centre exerting a downward pull upon the shoulder joint, destruction or severe injury of which would cause pointing deviation upward, is placed in the most superior and inner corner of the hemisphere, and includes adjacent surfaces of the superior and inferior semilunar lobes. The centre for outward tonus (shoulder joint) is located in the outer or lateral margin of the lobus semilunaris superior (3).

Of these centres the first two (those exerting inward tonus upon the wrist and shoulder movements respectively) are by far the most important from the standpoint of practical diagnosis for the reason that they are the centres most commonly involved in cerebellar lesions of otitic origin.

In cases of cerebellar disease in which spontaneous pointing deviation has been present, various joints have quite frequently been demonstrably involved,—*e.g.*, shoulder, elbow, wrist, hip, knee, ankle; also the neck and the trunk as a whole. A careful investigation of the different joints will therefore prove of academic interest, and in the case of small and slowly growing tumors may be of very practical diagnostic importance. I am inclined to believe, however, that in any case of otitic cerebellar abscess in which the arm and wrist show no spontaneous pointing deviation and in which the vestibular reactions are normal, little of practical advantage will be gained from the investigation of other joints.

Evidences of the Correct Localization of Centres for the Wrist and Shoulder (inward tonus).—As bearing upon this point, two more cases from Barany's reports may be cited.

Case II.—In a case operated upon in the Vienna General Hospital the cerebellar dura in front of the sigmoid sinus had been freely exposed. During the further course of the operation an instrument in the surgeon's hands slipped, entering the cerebellum through the middle inferior lobe near the flocculus. After recovery from the anæsthetic, his pointing accuracy was tested. The wrist corresponding to the side of the surgical mishap deviated strongly outward, the opposite hand and arm pointing accurately.

Case III.—Patient in this case presented certain general features of cerebellar abscess, but no focal symptoms. Exploratory operation was decided upon. First one cerebellum and then the other was explored, no abscess being found on either side. On each side an exploratory puncture was made through the slender lobe at the point located as the centre for the shoulder joint (inward tonus). The case therefore furnished a unique opportunity of corroboratory tests. After recovery from the anæsthetic, pointing tests were made. Both arms showed very marked and characteristic deviation outward.

Effect of Freezing.—As further evidence, Barany made use of several cases of patients who had recovered from operations leaving extensive dural areas in front of the sigmoid sinus uncovered, or covered only by a thin layer of skin. The experiment was repeatedly carried out by freezing by means of an ethyl chloride spray the centre for the shoulder (inward tonus). It has been found that after freezing this centre for a period of 2½ minutes, strong outward deviation has invariably been established.

While I have made use of Barany's cases to illustrate a set of phenomena the discovery of which was wholly the product of his brain and years of study, I have seen many cases in which these particularly useful focal symptoms were present. In one case which I saw with another surgeon, the patient who had been operated upon for mastoiditis, exhibited certain vague symptoms suggestive of intracranial involvement. A tentative diagnosis of "a low form of meningitis" had been made. The pointing tests showed well marked outward deviation confined to the right hand, and an operation the same day located and evacuated a right cerebellar abscess, the patient making a good recovery.

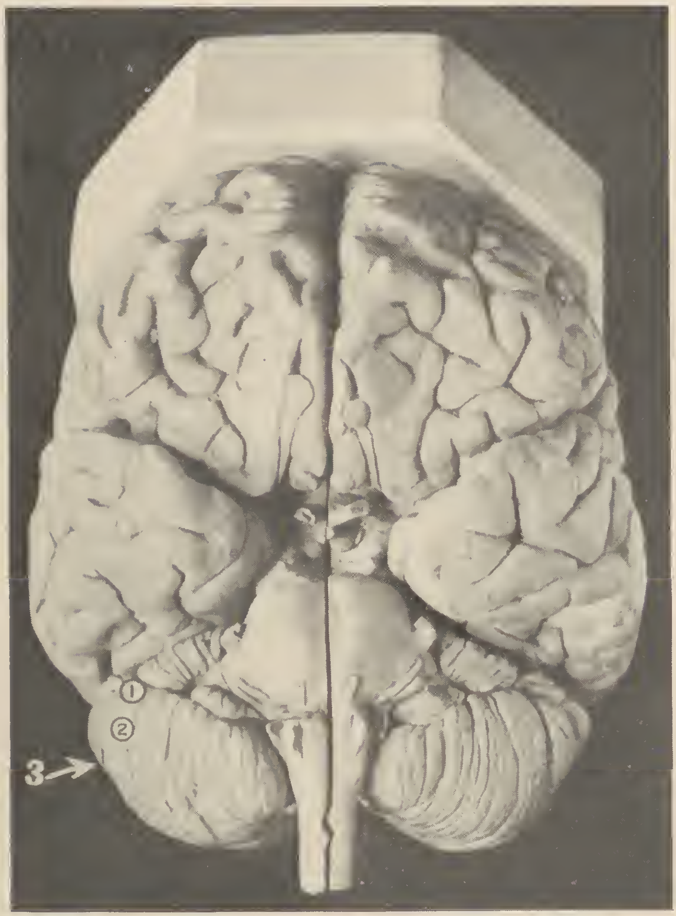


Fig. 196.—Inferior Surface of Encephalon. (1) Centre for inward tonus (wrist). (2) Centre for inward tonus (shoulder). (3) Indicates position on superior semilunar lobe of the centre for outward tonus, or pull, on the shoulder joint.

CHAPTER XV.

SURGICAL OPERATIONS FOR THE RELIEF OF SUPPURATIVE LESIONS OF THE MIDDLE EAR AND MASTOID PROCESS; MYRINGOTOMY; MASTOIDECTOMY; THE RADICAL OPERATION; OSSICULECTOMY.

MYRINGOTOMY; INCISION OF THE DRUM MEMBRANE; PARACENTESIS.

THERE is probably no one surgical procedure which has been the means of saving a larger number of lives than the judicious evacuation of pus from the tympanum through an incision of the drum membrane. It may be said to be positively indicated in every case of suppurative otitis media in which the drum membrane is found to be displaced outward (bulging) by pressure of confined pus. Reducing this statement to more exact terms, we recognize the following conditions calling for incision of the drum membrane:

FIG. 197.

FIG. 198.

FIG. 199.

FIG. 200.

FIG. 197.—Incision indicated in a suppurative lesion confined to the atrium.
FIG. 198.—Incision indicated in suppurative lesion involving the tympanic vault.
FIG. 199.—Incision supplementing inadequate perforation.
FIG. 200.—Incision occasionally indicated in chronic suppurative otitis media for the evacuation of pus retained in the vault.

1. Acute purulent otitis media confined to the atrium, as shown by bulging of the membrana tensa as a whole (Fig. 197).

2. Acute purulent otitis media involving the tympanic vault, as shown by bulging of Shrapnell's membrane and the upper posterior segment of the tense membrane (Fig. 198).

3. Suppurative otitis media with resolution delayed by the insufficient drainage provided by a small and inadequate perforation (Fig. 199).

4. Subacute otitis media characterized by an inflamed and infiltrated drum membrane and the presence of fluid—serum or pus—in the lower half of the atrium.

5. Acute exacerbation of chronic suppurative otitis media, with perforation or loss of membrana tensa and physical signs of pus retention in vault (Fig. 200).

Operative Dangers.—The surgical accidents occasionally reported as resulting from the abnormal position of important structures—*e.g.*, division of an exposed facial nerve, injury to an abnormally placed and exposed jugular bulb, etc.—are surgical dangers which are mentioned in most text-books. They depend upon exceedingly rare conditions which can not be allowed for by the physician. Of more serious import to the surgeon, because due solely to his error or carelessness, is the production of a surgical injury leading to suppurative labyrinthitis. Holinger has recorded a case in which the patient, suffering from acute purulent otitis media of the right ear, experienced severe dizziness immediately following a myringotomy. The vertigo and ataxia suggested labyrinthine disease and were so great as to confine him to bed. The symptoms of suppurative labyrinthitis rapidly gave place to those of meningitis, from which disease the patient died. Post-mortem examination showed fracture of both crura of the stapes and rupture of the capsular ligament, leaving the vestibule open to infection. To this case the writer can add one which came under his observation. The patient, a man of thirty whose hearing had previously been perfect, suffered an attack of acute middle-ear inflammation with the usual accompanying earache. He called upon a physician, who incised the drum membrane. Immediately following this operation, the patient experienced very severe dizziness and was obliged to go to bed. During the next ten days he was confined to bed by vertigo, nausea, and vomiting, at first severe but showing a gradual diminution. When I first saw him the drum membrane had healed but was still much congested. The right ear was absolutely deaf for all sounds. There was no caloric reaction. Apparently this was a case of traumatic infection of the labyrinth, from which the patient had recovered with absolute loss of function. Dr. John R. Page has informed me of a similar case which came under his personal observation. How often such accidents have actually occurred there is no means of determining. The important fact is that they do occur and that they are preventable. I am inclined to believe that the stapes or region of the oval window furnishes the usual point of attack, and that the injury is in most cases the result of a lack of exact knowledge or appreciation of tympanic anatomy rather than of a rough or careless use of instruments.

It will be remembered that neither the drum membrane nor the inner tympanic wall is in a plane at right angles to the long axis of the bony meatus. The drum membrane, for example, is very obliquely placed, forming decidedly obtuse angles with the roof and posterior wall of the bony meatus, and very acute angles with its floor and anterior wall. The

inner tympanic wall likewise is directed from above downward and strongly inward, so that it also forms an obtuse angle with the plane of the roof of the bony meatus and an acute angle with that of its floor (Fig. 201). These facts are restated and emphasized with the purpose of enforcing a point I wish to make,—viz., that the incision of the drum membrane should in every case begin below and be extended upward, and never in the reverse direction,—*i.e.*, beginning above and extending downward toward its lower margin.

Fig. 201.—Vertical section through external auditory canal and tympanic cavity.

It will be recalled that in incising an intact but bulging membrane the incision is practically always made through its posterior segment. If now we essay to make our incision from above downward, and plunge the knife through a point at or near its upper margin, with the cutting edge directed downward, we shall be likely to forget the normal slant of the membrane, and find later that the knife has quickly cut its way out, making only a short and totally inadequate incision (Fig. 202). Keeping the oblique plane of the drum membrane in mind, it is, of course, possible to obviate this error by carrying the knife simultaneously down-

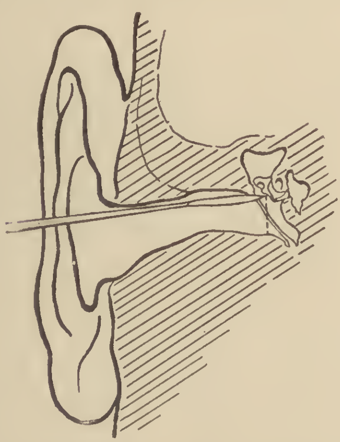


Fig. 202.—Diagram showing technical error likely to result from an incision made from above downward.

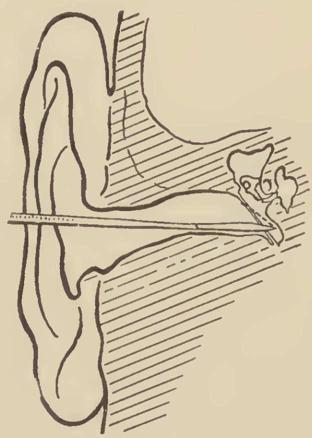


Fig. 203.—Diagram showing double movement (*i.e.* downward and inward) required in incising the membrane from above downward.

ward and inward, so that it emerges from the drum membrane only at its lower margin (Fig. 203). But in trying to execute this rather difficult

movement there is some danger that we may carry the point of the knife too far inward at the start, and, in the case of a low and prominent incudostapedial articulation, engage this or even the crura of the stapes, and with the downward stroke either drag the stapes altogether out of the oval window or at least produce a rupture of its capsular ligament, thereby admitting pus into the cavity of the vestibule (Fig. 204). If, on the other hand, we begin at the lower margin of the membrane and bear in mind its oblique plane, we can make the incision while withdrawing the knife.

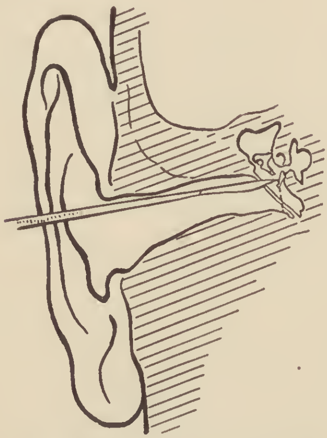


FIG. 204.—Dangerous accident (stapedial dislocation) which may result from an incision from above downward.

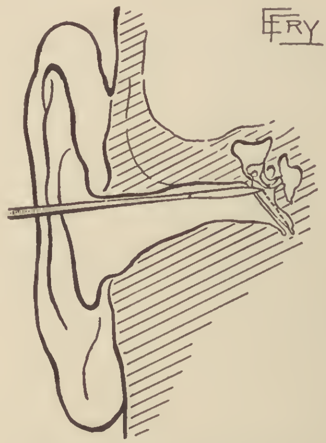


FIG. 205.—Incision of drum membrane from below upward, the correct method.

Cutting in this way, the point of the knife is moved constantly away from danger points; and with ordinary care we are in no great danger, even though we may touch the incudostapedial joint, of producing serious injury or traumatism (Fig. 205).

Injury of the Chorda Tympani Nerve.—This is an occasional accident resulting from an extensive incision of the drum membrane. It probably occurs oftener than either the physician or the aurist knows. It is announced altogether by subjective symptoms,—*i.e.*, the patient experiencing a sensation of numbness, and also losing the sense of taste on the side of the tongue corresponding to the ear incised. Whether the division of the nerve is usually complete or only partial, the resulting disturbance is, as a rule, short-lived. The symptoms referred to have occurred several times in the writer's experience, but have never seemed to disturb the patient greatly, and have always disappeared, or at least ceased to attract the patient's attention, within a few weeks.

PREPARATION FOR OPERATION.—The preparation of the patient is simple. The auricle if obviously unclean should be scrubbed with soap and water and dried. With the patient lying with the diseased ear directed

upward, the canal is filled with peroxide of hydrogen, which should be allowed to remain four or five minutes. The ear is then irrigated with a warm solution of bichloride of mercury, 1 in 2000, or carbolic acid, 1 in 200. A full quart of the solution should be allowed to flow in and out of the ear from a fountain syringe. The canal is then lightly packed with sterile gauze or cotton. This completes the preparation of the operative field.

The instruments, consisting of aural specula, cotton applicators, and bayonet forceps, are cleansed by boiling, the knife-blade being immersed first in strong carbolic acid and then placed in alcohol. As to the preparation of the hands, I prefer simply to wash them thoroughly with soap and water, then arrange my head-light or mirror, and at the last minute—*i.e.*, just as the patient is passing under the influence of the anæsthetic—to draw on a pair of previously sterilized rubber gloves.

Anæsthesia.—I believe very strongly that unless there are positive contra-indications the patient should be under the influence of a general anæsthetic. For this purpose nitrous oxide is an ideal drug. It acts quickly, is easily prolonged to the time required for a double myringotomy, —or longer if necessary,—and usually leaves the patient without any sense of discomfort referable to its administration. Aside from the saving of unnecessary pain to the patient, it enables the physician to make his incision with exactness and precision, which is not always possible without anæsthesia. In the case of children the use of a general anæsthetic is even more important, since, if it is omitted, the little patient may conceive a fear of the physician which will render all subsequent treatment most difficult.

Local Anæsthesia.—I know of no efficient local anæsthetic appropriate to this operation. A solution containing cocaine and carbolic acid in the strength of 10 per cent. of each is quite efficient in some cases, in others seeming to have comparatively little influence upon the pain. It is applied by saturating with the solution a small pledget of sterile absorbent cotton which is placed in contact with the drum membrane and allowed to remain not longer than two minutes. The use of stronger carbolic solutions has been advised, and naturally may be counted upon to act with greater certainty upon the sensory nerves of the part. But they are to be condemned as subjecting the drum membrane to very considerable risk in the destructive processes which not infrequently follow the local use of carbolic acid in strong solutions.

Technic.—Before attempting this delicate and very important little operation, the student should have a clear appreciation of the mechanical results which he wishes to achieve. We no longer speak of "puncturing" the drum membrane, because we now know that the most extensive incision which the lesion permits will be none too long for perfect drainage and, while affording better and more permanent relief of symptoms, will eventually heal quite as readily, and perhaps more perfectly, than a smaller opening.

Having determined upon the extent and direction of the incision, we should endeavor to carry this into effect without injury to other tympanic structures. In other words, if our aim is to drain the tympanum through

an opening in the drum membrane, we should endeavor to confine our incision to the drum membrane. The contention of Dench,[1] that we should incise not only the drum membrane but also the inflamed mucosa covering the inner wall, is open to the following objections,—viz., (1) To incise voluntarily a congested mucoperiosteal membrane covering the bony wall of a pus-filled cavity opens the way to possible pathways of infection which may lead to regions beyond our surgical control (*e.g.*, to labyrinth or meninges). (2) To cut with the knife-point in contact with the inner tympanic wall is to incur risks of injury to the labyrinthine capsule,—*e.g.*, stapes, capsular ligament, or at some point of erosion upon the promontory. Such an injury might easily lead to suppurative labyrinthitis.

It is assumed that the physician will have gained some facility in the manipulation of instruments,—*i.e.*, with probes, cotton applicators, etc.,—before attempting to use the myringotomy knife. The knife is most easily controlled if held between thumb, index and middle fingers, much as we hold a pen in writing (Fig. 206). The commonest types of failure are those

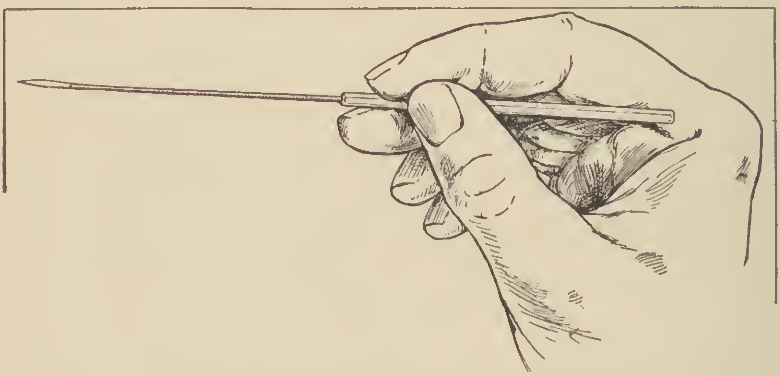


Fig. 206.—Correct method of holding myringotome.

in which the incision is either (a) not in the drum membrane, or (b) not so placed as to insure the most perfect drainage. Neither of these accidents is particularly uncommon with beginners. Failure to include any portion of the drum membrane in the incision is obviously due to the operator's failure to recognize normal landmarks or, these being obscured, failure to distinguish between the bulging drum-head and the contiguous swollen canal wall, so that on his next inspection of the ear he finds the drum membrane still bulging or spontaneously ruptured, and is able to distinguish the line of his incision in the posterior wall of the canal. This mortifying experience has occurred to many a beginner in otology and is perhaps a wholesome one in bringing home to him the importance of a careful study of the small operative field before using the knife. This particular accident

[1] Dench: Diseases of the Ear, 1904, p. 487.

is likely to occur only in those cases in which the bulging posterior segment of the drum-head merges without any distinguishable line of demarcation into the swollen posterior canal. It is avoided by a careful search for the hammer handle, which, though frequently obscured, can usually be located by a slight linear depression along the line of its attachment to the drum membrane. Having recognized, or at least determined the position of the hammer handle, our incision should in these cases be not more than 1 or $1\frac{1}{2}$ mm. behind it.

The second and commonest error—*i.e.*, of an improperly placed incision—is probably more often due to imperfect control of the knife than to a lack of knowledge as to where the incision should be made. I believe that these failures are oftenest explained by the surgeon's failure to introduce the knife at the correct point upon the drum membrane. He sees the point which he wishes to represent his initial puncture, but fails to strike it with the point of his knife. The cause of this deviation is probably analogous in many cases to swerving of a pistol in the hands of a beginner at the time he pulls the trigger. Once the knife is improperly introduced, the remainder of the incision is necessarily thrown out of its correct course, though the operator may be blinded to this defect by the resulting hemorrhage until the patient has come from under the influence of the anæsthetic. If the operation has for any reason been performed without the aid of a general anæsthetic, it will be usually quite impossible to correct any error in one's initial incision. It is expecting too much of the patient to require him to endure the torture of a second incision in so exquisitely sensitive a structure as the inflamed drum membrane.

It will be seen from the above that the author has endeavored to suggest the commoner types of failure, their causes, and the means by which they may be avoided, rather than to describe in detail the steps of the operation. Assuming that the physician possesses a practical knowledge of the drum membrane and its tympanic relations, and that he knows where the incision should be made, I believe that there are only three essentials to his acquiring quickly a satisfactory technic in this very important little operation,—viz., (1) he should insist upon having his patient under the influence of a general anæsthetic; (2) he should carefully and without haste bring the point of his knife, with cutting edge directed upward, to the point which he wishes to represent the lower end of the incision; and (3) he should extend the incision upward without haste and always under the guidance of sight,—*i.e.*, without at any moment losing sight of the line of his incision.

Removal of Post-operative Clot.—Following a myringotomy there is always a moderate hemorrhage into the canal, and, unless this receives attention, the purpose of the operation may be defeated by the resulting clot. I, personally, like to wait about ten minutes and then syringe the ear with a warm solution so as to see for myself the expulsion of the coagulating blood. If the first irrigation is too long delayed and then imperfectly carried out by the nurse or attendant, it is possible that the clot may

not be dislodged and may partially or completely seal the incision, preventing the drainage of the infected cavity which was the chief end in view. I have known this actually to occur. The initial clot having been removed, there is usually no recurrence.

Possible Repetitions.—It must be remembered that one of the characteristic features of the normal drum membrane is its remarkable tendency to rapid healing after incision or accidental puncture, provided infection of the tympanic cavity does not take place. This tendency occasionally acts disadvantageously in cases of purulent otitis media, quick partial healing interfering with the free drainage which the incision was intended to secure. When this occurs, there should be no hesitation as to repeating the operation. I have known cases in which the judicious repetition of this lesser operation has undoubtedly saved the patient from the severer ordeal of a mastoid operation.

SURGICAL LANDMARKS OF THE MASTOID CORTEX.

Probably in no region of the body outside of the skull do so many important structures so nearly approach each other as in the body of the temporal bone. Here, within a space little more than half an inch square, are found the essential organ of hearing, the semicircular canal system and vestibular apparatus, the cochlear and vestibular branches of the auditory nerve, and the horizontal portion of the facial nerve. Injury to any one of these structures must inevitably cause either grave functional disorder or actual deformity. A little more than half an inch from any of them, we come upon the thin bony coverings of the sigmoid sinus, the bulb of the jugular vein, the brain, or the cerebellum. Clearly it is a region in which the surgeon should know his ground.

There are two sets of surgical landmarks with which it will repay the student to become familiar before attempting to operate upon the living subject,—viz., (1) those upon the outer surface of the mastoid cortex which enable him to determine approximately the positions of important structures beneath; and (2) those encountered within the bone during operation which indicate the exact location of structures he may wish either to attack or to avoid.

Before describing the various operations, it may be worth while to consider briefly the surgical landmarks of the outer surface of the mastoid (Fig. 207).

The Suprameatal Spine (*Henle's Spine*).—At the point where the upper posterior wall of the bony meatus terminates or merges into the outer surface of the mastoid, is a crescentic bony spine (Fig. 207, *a*) known as the suprameatal spine, or spine of Henle. It is absent in the new-born, but is usually developed early in child life and is a very constant landmark in the adult. The centre of this spine marks fairly accurately—passing from without directly inward—the position of the floor of the *aditus ad antrum*.

It will be recalled that the antrum and tympanic vault are in reality but a single cavity, the antrum representing the posterior end of the tympanic vault. The division of the antrotympanic cavity into two portions—*i.e.*, the vault or attic in front and the antrum behind—is brought about by a central constriction known as the aditus ad antrum. The aditus is,

FIG. 207.—Surgical landmarks of mastoid cortex: *a*, spine of Henle; *b*, posterior root of zygoma; *g*, temporal ridge; *c–d*, line representing direction of horizontal portion of facial canal; *e–f*, line indicating course of descending portion of facial canal.

therefore, not a narrow passage, as described in some of the earlier textbooks, but a triangular space of considerable size. The position of the floor of the aditus in relation to the outer mastoid cortex is indicated by the centre of Henle's spine. This surgical guide is of practical value in certain cases in which the antrum is abnormally small.

Posterior Root of Zygoma.—Beginning anteriorly in the sharp upper edge of the zygoma, and running backward and usually more or less upward upon the squama, is a usually well-defined ridge, which forms part of the temporal ridge. That part of the temporal ridge which is just above the bony meatus and spine of Henle is known as the posterior root of the zygoma (Fig. 207, *b*).

In mastoid surgery the posterior root of the zygoma is a useful and important landmark as denoting the level of safety above which one may in certain temporal bones enter not the antrum, but the middle fossa of the skull. The rule, therefore, should be to begin one's initial opening well forward,—*i.e.*, a little behind the spine of Henle,—and below the level of the posterior root of the zygoma. In observing this rule, one must bear

in mind that the temporal ridge often curves upward upon the squama to a level considerably above that of the floor of the mid-cranial fossa. The level of safety is illustrated in Fig. 208 by the dotted line *a–b*, between which and the temporal ridge is a triangular space (*c*), through which one might easily enter the brain cavity.

Fig. 208.—Level of safety in opening mastoid. The line *a–b* is a horizontal extension backward of the posterior root of the zygoma. Between this line and the posterior upward extension of the temporal ridge is found in many bones a point, *c*, through which the chisel would enter not the interior of the mastoid, but the mid-cranial cavity. The line *a–b* represents, therefore, the level of safety.

Course of the Horizontal and Vertical Portions of the Facial Canal.—While the facial nerve is deeply situated, and therefore liable to exposure and direct injury only in the depth of an operative wound, it is to some extent subject to injury by concussion or transmitted force, and may therefore be endangered by careless surgery in the simplest operation upon the mastoid.

The "horizontal" and "vertical" portions of the facial canal are not exactly horizontal and vertical in direction. If we will imagine a line, more or less horizontal in direction, which passes through the centre of the spine of Henle, its anterior extension forming an acute angle of about 15 degrees with the upper margin of the zygoma (Fig. 207, *c–d*), this will indicate fairly accurately the direction upon the inner tympanic wall of the horizontal portion of the facial canal. Again, imagine a line, approaching the vertical in direction, which passes through the centre of the supra-meatal spine above and is in contact with the anterior margin of the mastoid below (Fig. 207, *e–f*), and we shall have a fairly accurate guide to the course of the descending portion of the facial canal, from its bend, or knee, just beneath the floor of the aditus to its termination in the stylo-mastoid foramen.

Fig. 210.—Instruments called for in any surgical operation.

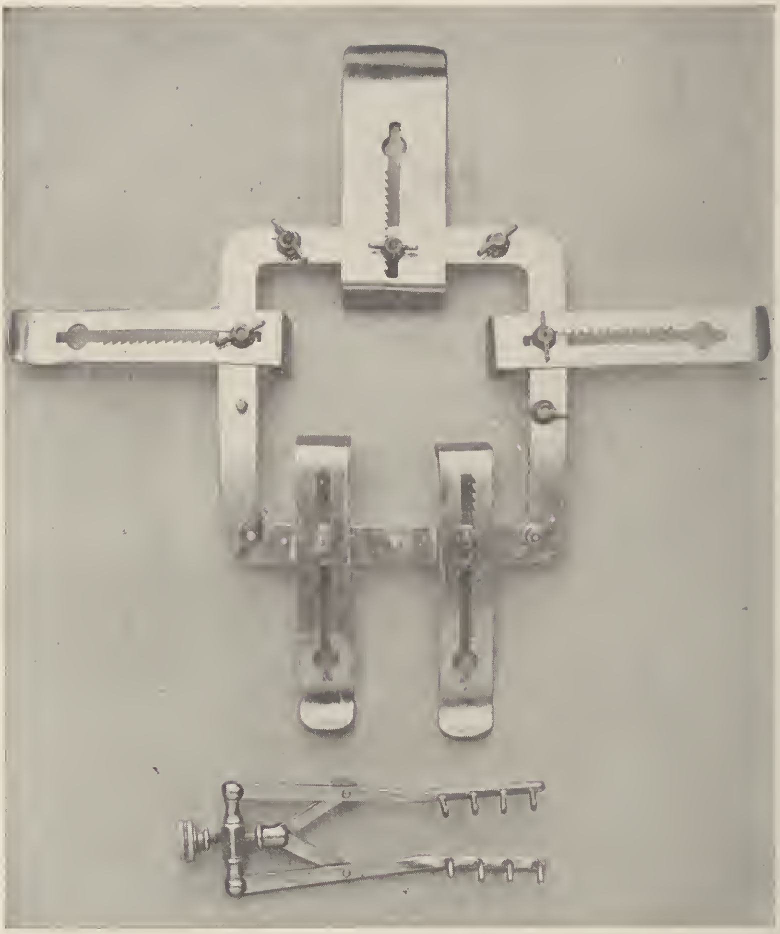
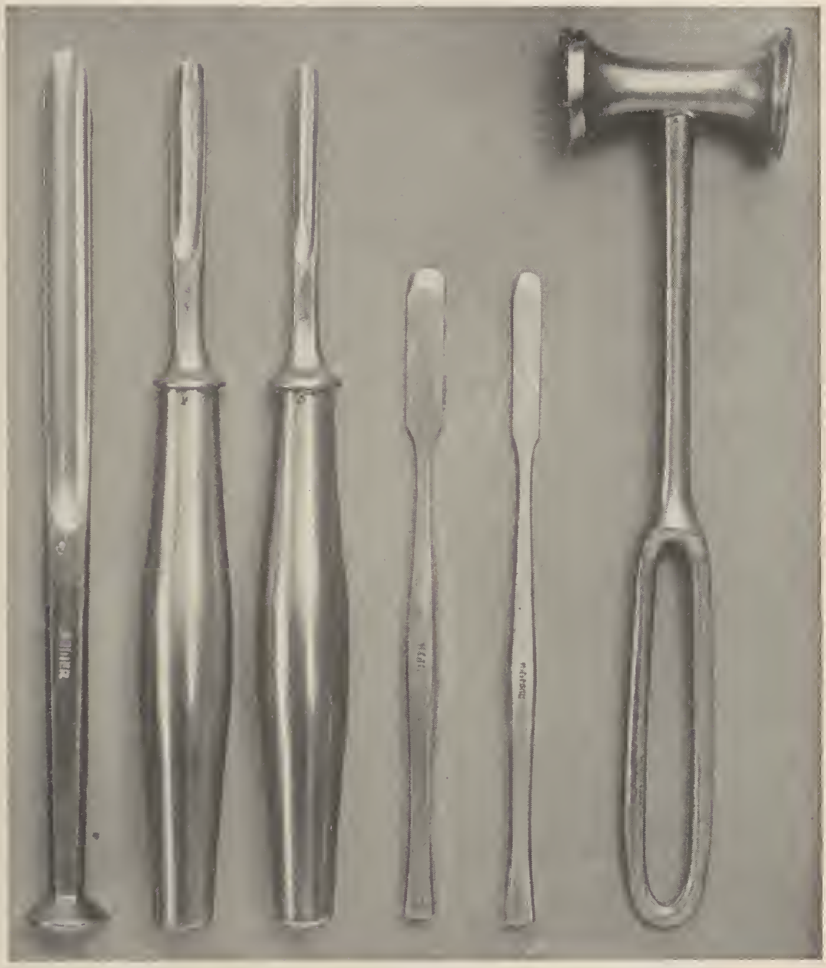


Fig. 211.—Self-retaining retractors. *a*, Kerrison's retractor; *b*, Allport's retractor.

Fig. 212.—Langenbeck's periosteal elevator.

A B C D E F



A–Fig. 213.—Large grooved chisel, or gouge.
B–Fig. 214.—Whiting's gouge.
C–Fig. 215.—Whiting's gouge.
D–Fig. 216.—Jansen's chisel.
E–Fig. 217 —Jansen's chisel.
F–Fig. 218.—Mastoid mallet.

FIG. 219.—Mastoid rongeurs

MASTOIDECTOMY; THE MASTOID OPERATION; SOMETIMES CALLED "SCHWARTZE'S OPERATION."

For a chronological account of the gradual development of this important operation, the reader is referred to the historical sketch in Whiting's book on the mastoid operation.[2] For practical purposes it will suffice here to record briefly the succeeding steps in the evolution of the operation as practised to-day. (1) The first operations for the relief of suppurative mastoiditis of which we have authentic accounts were performed in the early half of the eighteenth century, and were undertaken chiefly for the evacuation of confined pus. The surgeon therefore contented himself with drilling a hole through the mastoid cortex; and, as this provided neither adequate drainage nor removal of diseased bone, a majority of patients died, and the operation fell justly into ill repute. (2) The next operation, still in the nature of a trephining operation, aimed to provide freer drainage and incidentally to remove such bone as was obviously diseased. This marked an advance, but still failed by its results to establish the procedure as one of universally recognized usefulness. (3) Finally, in 1873, Schwartze[3] advocated and described a systematic method of removing the mastoid cortex, breaking down the mastoid cells, and providing at once free drainage from all infected spaces within the mastoid. This work of Schwartze laid the foundation upon which has been built up our modern system of aural surgery, and explains the fact that the simple mastoid operation is often spoken of as "Schwartze's operation."

FIG. 209.—Area shaved for mastoid operation.

PREPARATION OF THE PATIENT.—The side of the head should be shaved over an area measuring fully three inches in all directions from the auditory meatus of the diseased ear (Fig. 209). The meatus should be irrigated with a solution of bichloride of mercury, 1 in 2000, and lightly packed with sterile gauze. The parts are then scrubbed with soap and water, thoroughly dried, and a covering of sterile gauze applied. These preliminary steps should

[2] Whiting: Modern Mastoid Operation, Blakiston, pp. 13 to 29.
[3] Schwartze: Arch. für Ohrenh., Bd. ii, 1873.

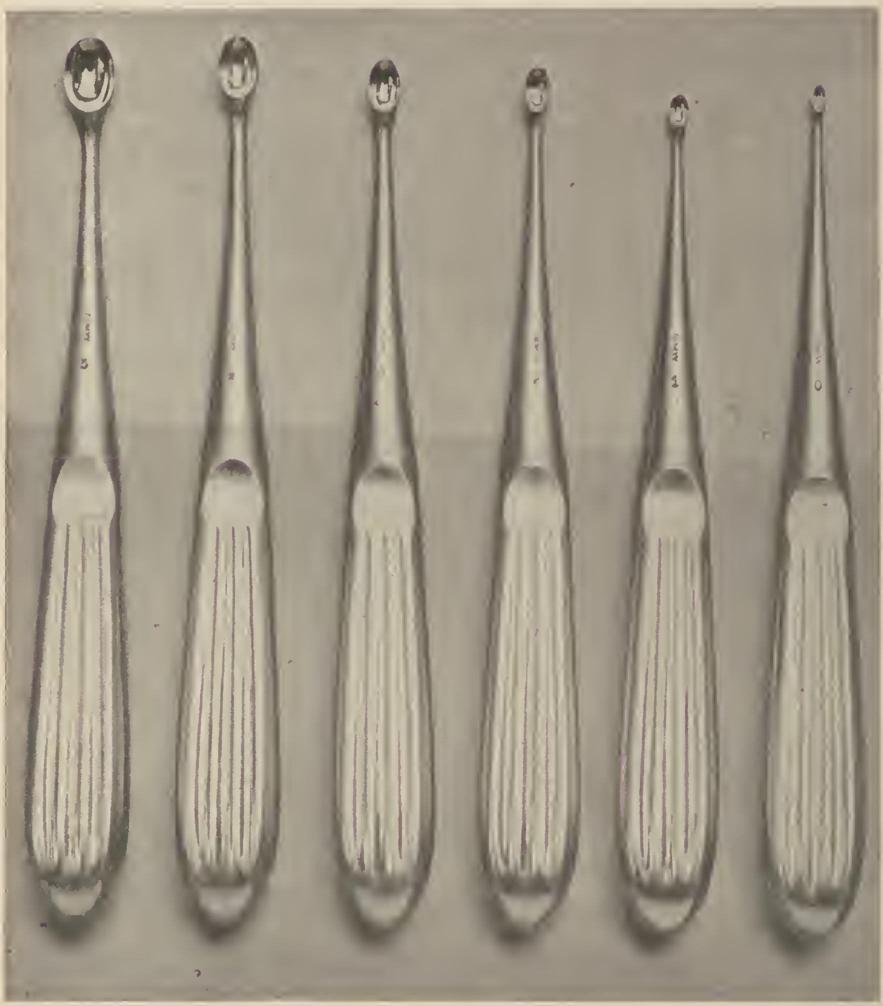


FIG. 220.—Mastoid curettes.

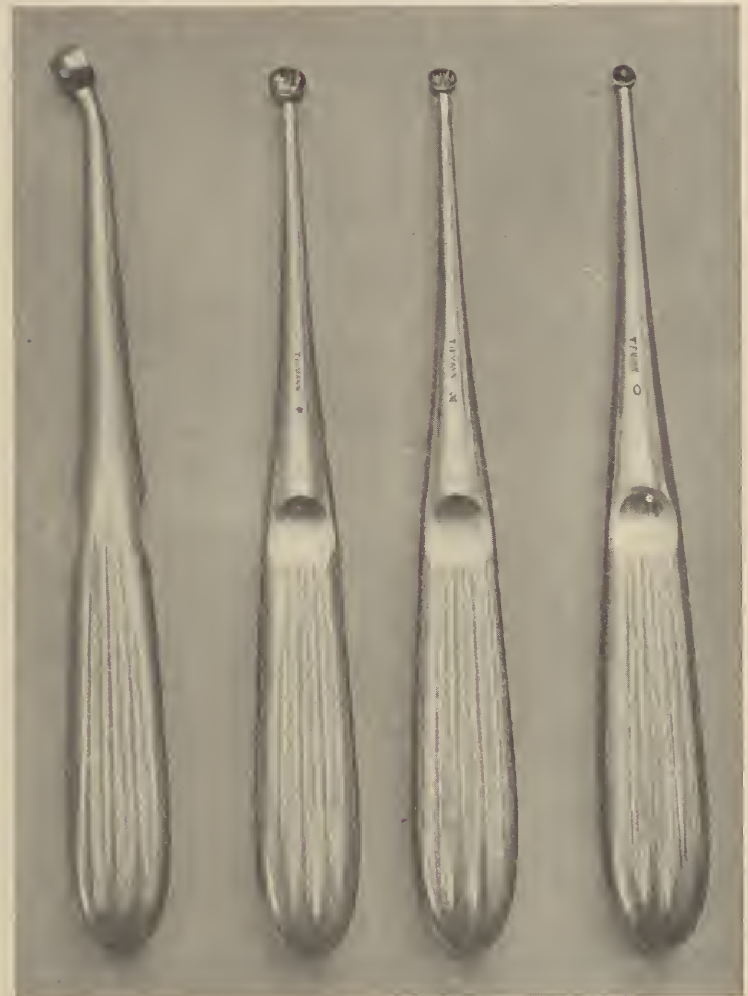


Fig. 221.—Richards's mastoid curettes.

precede the operation by several hours. When the patient is on the table and under the influence of the anæsthetic, the shaved area and the auricle, both anterior and posterior surfaces, are painted with tincture of iodine. This provides rapid and perfect sterilization. A common practice is to wipe the painted surfaces, just before the initial incision, with sterile cotton soaked in alcohol. This removes the iodine stain and perhaps lessens the risk of skin irritation, but whether it is wise, *i. e.*, from the viewpoint of dependable asepsis, is open to question.

When tincture of iodine is to be employed in an emergency case in which all the preparations are made when the patient is on the table, the shaved area should be rubbed vigorously with dry cloths, and the tincture of iodine immediately painted thereon. Any superfluous use of water and antiseptic solutions just before the iodine is applied is known to interfere with the bactericidal action of the latter. The iodine alone provides complete sterilization.

The surgeon's hands are prepared in accordance with general surgical rules which need not be stated here. In addition to a very thorough cleansing with soap, water, and brush, and immersion for a minute or two in 1 in 2000 bichloride solution, the writer believes that there is distinct advantage in the use of rubber gloves which can be subjected to more drastic sterilization than can possibly be applied to the hands.

Instruments.—The instruments required for a mastoid operation may for convenience be thought of as belonging to two groups,—viz., (1) *those in common use in any operation,—e.g.*, scalpels, artery-clamps, thumb forceps, scissors, ordinary retractors, needle-holders, etc.,—and (2) *instruments especially designed for mastoid or bone surgery.* It is so important that these latter should be of correct design that the writer has had cuts made from photographs of those which in his own practical experience have proved most satisfactory.

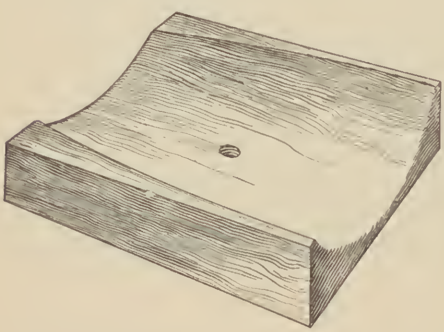


Fig. 222.—Wooden head-rest, or block, for use in mastoid surgery.

In addition to the above, it is always well to have a set of ear specula, cotton applicators, and a myringotome in readiness, as it is frequently necessary to incise the drum membrane at the time of the mastoid operation; and it is only by including these tympanic tools among our mastoid instruments that we can be sure of having them when they are required.

Position of Patient on Table.—The patient lies on his back, with head turned so that the diseased ear is directed upward. The head rests upon a sandbag or, better, upon a specially-designed wooden head-rest (Fig. 222), which is wrapped in sterile towels, and which prevents unnec-

essary movement. The towels about the head are so arranged as to leave the face fully exposed to the view of the anæsthetist, who sits at the side of the table to which the face is turned. *The anæsthetist should watch for unilateral facial twitching, which usually announces any surgical injury or even irritation of the facial nerve.* Such danger signals should be at once reported to the surgeon.

The Initial Incision (Fig. 223).—The initial incision should begin at, or just below, the tip of the mastoid (*b*) and be carried upward, parallel with, and one-quarter of an inch behind, the posterior line of auricular

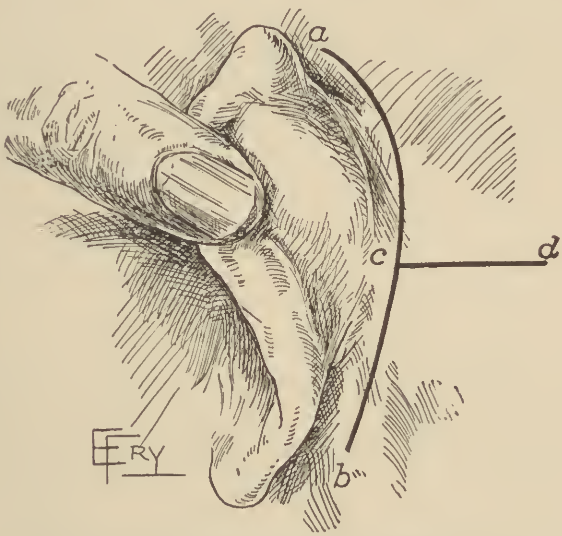


Fig. 223.—Initial incisions for exposing the mastoid cortex.

attachment, to end at a point above the upper attachment of the pinna (*a*). In its lower two-thirds it should divide all structures down to the bone, but in the upper third the pressure should be reduced so as not to include the temporal muscle. While many surgeons divide the muscle throughout, it is rarely necessary to do so. If from the temporal ridge, to which the muscle is attached, the tissues are divided only down to the surface of the muscle, the anterior skin flap can be easily deflected from it so as to permit the necessary displacement forward. By attention to this point, we materially reduce the initial hemorrhage, and avoid unnecessary stripping of the periosteum from the superior plate of the squama, a region not necessarily involved in the bone operation. Should it subsequently become necessary to do so, it will be found an easy matter to separate the muscle from its lower attachment and retract it upward.

The hemorrhage following the initial incision is always more or less free, the chief bleeding points being (a) from the temporal muscle when it is cut, (b) from the sternomastoid fibres at the tip, and (c), between

these points, from the divided periosteum rather than from the overlying tissues. When the initial bleeding is not excessive, the surgeon will save time by ignoring it until he has elevated the flaps anteriorly and posteriorly, when the chief bleeding points will be brought into better view, and many of the smaller ones will have ceased spontaneously.

In deflecting the flaps, care should be taken to elevate the periosteum without unnecessary laceration or traumatism. This can be most easily accomplished by means of Langenbeck's elevator (Fig. 212). With its bevelled edge placed firmly upon the bone and parallel with the edge of the incised periosteum, the latter can be elevated without laceration throughout the greater part of the incision. With practice this part of the operation can be done very quickly,—*i.e.*, from a few moments to two or three minutes, according to the roughness of the cortex and the strength and thickness of the tendinous attachment. Naturally the most difficult part of the cortex to uncover is found just above the tip where we come upon the tough tendinous fibres of the sternomastoid muscle. These can in some cases be cleanly elevated from the bone by very careful use of the elevator, but in muscular subjects it is usually necessary to cut them away with the points of a pair of curved scissors.

When the tip has been properly cleared, the anterior flap pushed forward so as to expose the suprameatal spine, and the posterior flap separated from the bone to a distance of about an inch, we have an exposure which in many cases is quite adequate to the requirements of the operation. There are cases, however, in which it is not. As soon, therefore, as it becomes evident that the exposure of additional bone will facilitate the operation, this can and should be obtained by the supplementary incision suggested by Whiting. Starting opposite the orifice of the bony meatus, this runs backward roughly at right angles to the initial incision (Fig. 223, *c-d*), thus dividing the crescentic posterior lip of the wound into two triangular flaps. These, properly deflected, usually provide easy access to all parts of the diseased mastoid (Fig. 224).

The Bone Operation.—In attempting to present clearly the essential points of this important operation, I shall depend very largely upon a series of illustrations representing its successive steps.

I. *The initial opening* into the mastoid is made by the removal of a strip of cortex whose posterior boundary is indicated by the heavy dotted lines in Fig. 224. It begins well forward,—*i.e.*, from 4 to 5 mm. behind the suprameatal spine,—its anterior boundary closely following the posterior canal wall and anterior margin of the mastoid to the tip. For removing the cortex, the safest instrument is a gouge of medium size (Fig. 214). It should be held very obliquely, so as to remove a layer of bone not more than one-eighth of an inch in thickness.

If we imagine a horizontal line tangent to the roof of the bony meatus and a vertical line tangent to its posterior wall, it is clear that these lines will cross each other just above and behind the spine of Henle (Fig. 224). Just behind their point of crossing is the point at which the gouge should enter the bone (x). It should be propelled by very moderate blows of the

mallet, and carefully guided along the anterior margin to the tip. Used in this way, even if the cortex be thin, and the sigmoid sinus situated far forward and near the surface, the latter is not likely to be injured, whereas, if the gouge be held more at right angles to the cortex and made to pene-

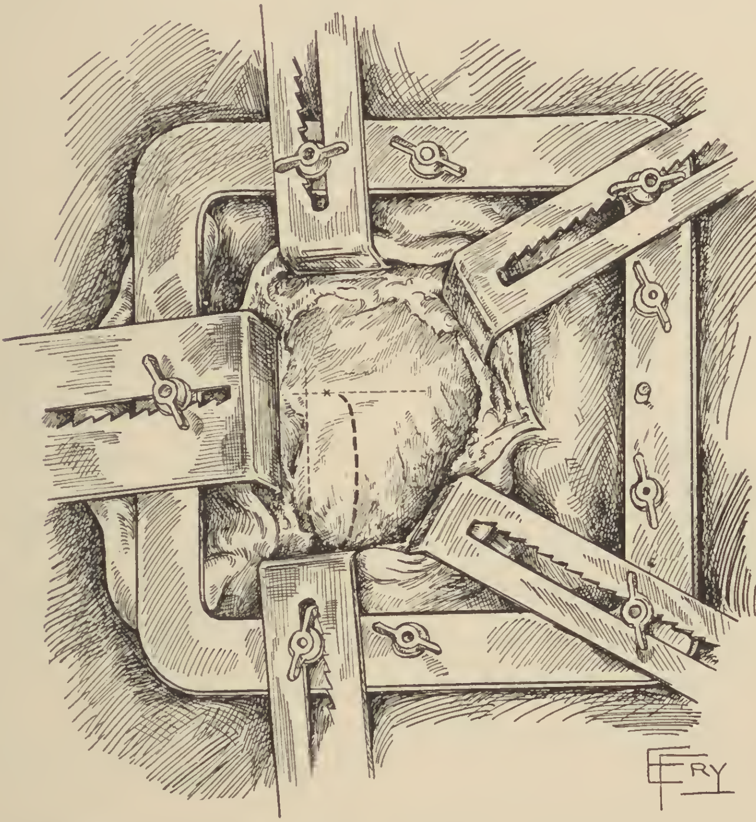


Fig. 224.—Mastoid cortex adequately exposed for operation. x indicates point at which gouge should enter bone.

trate more deeply into the unexplored bone, there is distinct danger of opening the sinus, with resulting profuse hemorrhage and the grave possibility of serious intrasinous infection. Ordinarily—*i.e.*, when the mastoid surface is free from unusual depressions—this anterior strip of bone may be removed with a single continuous excursion of the gouge. When the cortex is thin, the mastoid cavity is usually opened with the first layer of bone removed, but a thick cortex we may have to remove in successive layers. The opening thus formed may then be widened by the removal of a second strip (Fig. 225). This enlarged opening discloses the diploic or pneumatic structures, according to the character of the bone. After this

stage is reached, the writer usually makes no further use of the mallet or gouge, preferring to substitute other instruments which do their work without the jar, or concussion, incident to the use of the mallet.

NOTE.—The advantage of the gouge over the chisel lies in the fact that the former simultaneously excavates and removes a shallow continuous layer of bone, whereas with the chisel one must outline the edges of small areas of bone which are then prised or lifted from their position. This is a slower process which is also attended with greater possibilities of injury to important structures below the cortex.

II. *The second step* is the removal of the diploic or intercellular bone substance now exposed in the anterior part of the mastoid. This is most safely accomplished by means of a bone curette of medium size. In order

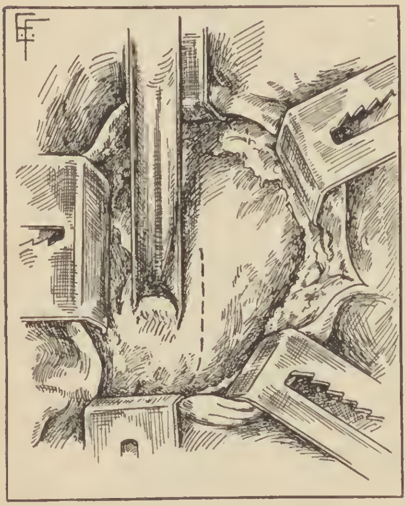


FIG. 225.—Position of gouge in removing mastoid cortex. Dotted lines indicate posterior boundary of second strip of cortex removed.

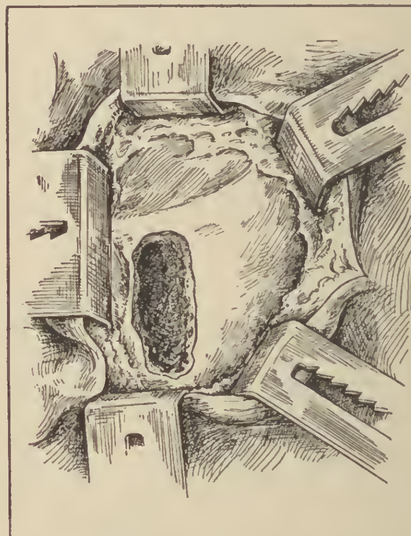


FIG. 226.—Representing the extent and outline of the initial opening usually made with the mallet and gouge.

to avoid risk of injury to the sinus, the position of which we may not as yet know, the curette should at first be used with great care and with its cutting edge directed slightly forward toward the anterior plate of the mastoid. In this way a deep groove is made, which hugs the anterior wall of the mastoid and is gradually deepened until the firm, harder bone representing the floor of the general mastoid cavity is reached (Fig. 226). If the groove of the sigmoid sinus traverses this part of the mastoid, its arched bony plate will be detected by its harder, smoother surface; or it may be that the vessel itself may be exposed by the inadvertent removal at some point of its bony covering. This accident, while better avoided, need not cause anxiety beyond making us careful to avoid injuring its dural covering during the subsequent steps of the operation.

III. *Further Removal of Cortex.*—The next logical step is the removal of the remainder of the mastoid cortex. Having reached the stage shown in Fig. 226, we know that we shall have to further uncover the mastoid at least to the extent roughly shown by the dotted lines in Fig. 227. It is better to adopt a regular system in doing this,—starting either above and going downward toward the tip, or *vice versa*. Personally, I prefer to begin by removing the outer cortex of the tip, following this upward and backward, and finally removing the outer wall of the antrum. Undoubtedly the best instrument for this work is a rongeur of appropriate size and design. I prefer a rather large rongeur for working backward toward the posterior margin of the mastoid, and a smaller instrument for work about the tip and antrum. The advantages of the rongeur over the gouge or chisel are (1) absence of the jar or concussion which is inseparable from the use of mallet and chisel, a consideration of no little importance in some cases; and (2) the combined rapidity and safety with which it enables one to work. The one danger of the rongeur is the possibility of introducing the under blade too deeply or too far beneath an overhanging shelf of bone, and thereby including in its bite the membranous wall of the sinus. This, however, has never occurred in the writer's experience, and with ordinary care can be easily avoided.

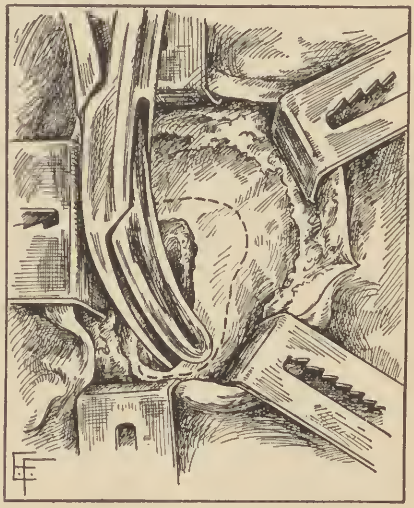


Fig. 227.—Illustrating the use of the rongeur in removing the mastoid cortex.

IV. *Treatment of the Tip.*—With most American aurists it has become an axiom that in every case the tip should be removed. This dogma has long seemed to the writer to require some modification, or at least a clearer definition than is vouchsafed in most text-books. Does removing the tip mean that we shall in every case expose the posterior belly of the digastric muscle by removing the inner plate of the tip? Personally I do not believe that it is always either necessary or advisable to do so. Unquestionably the tip cell or cells should be uncovered by removal of the external cortex, and the diploic or intercellular bone substance should be removed. If the inner plate thus exposed is diseased or so thin as to leave its nutrition in doubt, it also should be removed. But if the exposed inner plate is composed of comparatively thick, healthy bone, I can see no valid reason for its removal, nor have I seen one advanced. On the contrary, by leaving it unmolested, we avoid useless injury of the digastric muscle and lessen the danger of secondary infection at this point. In such a case its

lower margin should be rubbed smooth by means of a curette, but such part of the inner plate as is obviously sound and capable of maintaining its own nutrition should not be removed.

V. *Locating the Antrum.*—Ordinarily there is no difficulty in locating the antrum. Situated at a variable depth from the surface, and at a point just above and behind the posterosuperior wall of the bony meatus, it is found at the upper end of the original groove, or excavation (Fig. 226). Its identity may, if necessary, be confirmed by introducing a bent probe (Fig. 228), and passing its curved end forward through the so-called aditus ad antrum into the tympanic vault. If the end of the probe encounters no opening leading forward into the vault, we know that we have not entered the antrum.

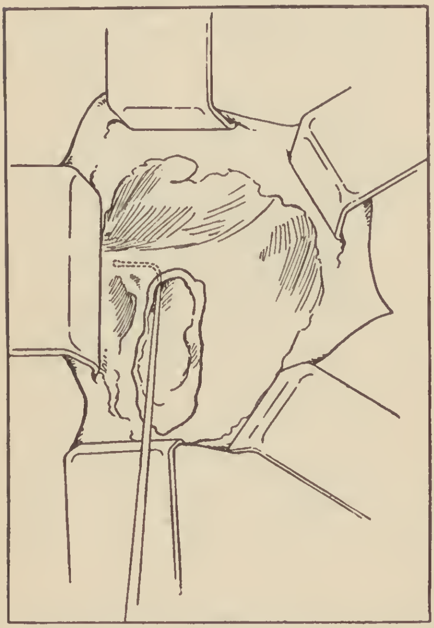


Fig. 228.—Diagram showing the use of the bent probe in locating the aditus.

If the antrum is small, beginners occasionally have difficulty in reaching it because the excavation is either (a) not deep enough or (b) has not been extended sufficiently in a direction upward and forward.

In bones of semisclerotic type the antrum is often exceedingly small, in some cases extending little if any beyond the limits of the space usually spoken of as the aditus. When in such a case the surgeon fails to locate the antrum in the situation in which he expects to find it, he may proceed to trace it in the following way: Pushing the anterior flap and auricle well forward, let him carefully note the position of the suprameatal spine and level of the posterior root of the zygoma (Fig. 229). Taking now two imaginary lines,—one horizontal and passing through the posterior root of the zygoma (*a–b*), and the other oblique and passing through the anterior and posterior ends of the suprameatal spine (*c–d*),—he has the angle, *b–e–d*. An arrow pointing forward and somewhat upward and bisecting this angle will necessarily point directly to the aditus, which may represent the only antrum present. As a supplementary guide, the surgeon may utilize the superior plate or roof of the mastoid process, following this forward to the antrum or aditus, of which its continuation forms the roof.

When for any reason there is difficulty in locating the antrum, the use

of these lines not only provides the correct point of attack, but in the case of a low-lying tegmen antri insures against the possible error of entering the middle fossa, and perhaps perforating the dura and brain, under the impression that the curette has entered the antrum.

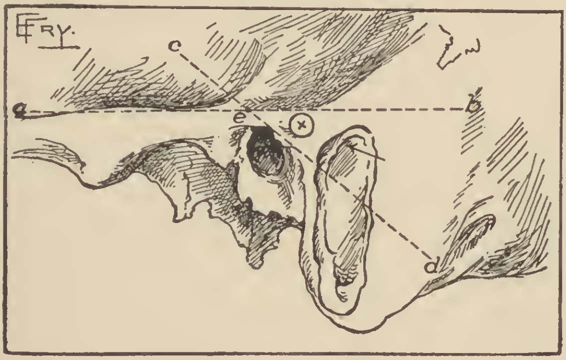


FIG. 229.—Lines representing surgical guides to a very small antrum.

VI. *Surgical Relations of the Aditus.*—It is often found, after the antrum has been cleared of pus and granulations, that the aditus is also filled with unhealthy granulations which it is our duty to remove. In using the curette for this purpose, the surgeon should bear in mind the relation of this space to the facial canal and horizontal semicircular canal (Fig. 230).

Within the tympanum, or rather upon the inner tympanic wall, the prominence of the horizontal semicircular canal is seen immediately above the horizontal portions of the facial canal (see Fig. 15 on page 11). As they extend backward, however, the facial canal passes beneath the floor of the aditus and there bends downward, from which point it passes more or less vertically to its termination in the stylomastoid foramen. The semicircular canal, on the other hand, being in the horizontal plane and slightly above the facial canal, curves backward and lies behind the aditus (Fig. 230). In curetting the walls of the aditus, therefore, we must remember that beneath its floor is the bend of the facial nerve, while just behind its posterior wall is one aspect of the horizontal semicircular canal. From this the surgical deduction is clear that, while we may use the curette with freedom against the anterior wall and roof, it should be employed only with great care and gentleness against the posterior wall and floor. The surgeon should always direct the anæsthetist to watch the face closely for unilateral facial spasm while he is working within the aditus.

Surgical Treatment of the Aditus.—Usually the tympanic lesion sufficiently antedates the mastoid symptoms to make this region a seat of advanced pathologic change. Frequently the mucosa is eroded and partly replaced by exuberant granulations. If these granulations are only partially removed from the aditus, what remains may shrink and the

parts rapidly regain their normal character. On the other hand, a small amount of granulation tissue left in this situation may mark the site of a necrotic area in the underlying bone. Such a focus of disease, if not surgically removed, may perpetuate the tympanic lesion. There is no question that many trying cases of delayed repair and some failures have been due to this cause.

Fig. 230.—Vertical section through the aditus.

Granulations attached to the roof and upper part of the posterior wall may be quickly and radically dealt with. Frequently a considerable mass of diploic tissue is attached to tegmen tympani, thorough removal of which provides a safer postoperative condition.

Granulations occupying the floor of the aditus are frequently attached also to the short process and body of the incus, a fact which should be borne in mind since considerable care is necessary in some cases to avoid dislocation or even extraction of the incus. Particularly in operating on young children is this danger a real one. Removal of granulations in this situation often brings the incus into view, and sometimes also the head of the malleus. In cases of long standing tympanic suppuration, which have not reached the stage characterized as chronic suppuration, exposure of one or both ossicles by careful removal of the outer wall of the vault may be necessary in order to bring the parts into a condition favorable to prompt tympanic repair. This, however, is exceptional. The contention of some surgeons that the incus should be exposed to view as a routine practice in all mastoid operations does not appeal to me as well founded.

VII. *Outlining of Sigmoid Groove; Angle between It and Mastoid Roof.*—In removing the diploic or pneumatic structures from the interior of the mastoid process, we reach in most directions the smooth bone of its limiting plates. Anteriorly we come upon the posterior canal wall and premastoid plate; above, upon the inner plate of the tegmen antri; posteriorly and at a distance from an eighth of an inch (exceptional) to a half inch or more from the posterior canal wall, upon the firm arched plate of bone forming the osseous covering of the sigmoid sinus. Internal to and in front of the sinus groove is the plate separating the mastoid cavity from the posterior fossa of the skull and the cerebellum. The modern conception of the requirements of the mastoid operation is that all these structures

should be outlined by the fairly complete removal of the intercellular bone substance. This is best done by means of a curette. For this purpose a large spoon curette is a safer instrument than a small one. Generally speaking, it is safer for the stroke, or sweep, of the curette to be up or down—*i.e.*, from tip to roof or *vice versa*—rather than from before backward. When the sinus groove is encountered, its outline is traced by removing the diploic tissue from it and its immediate neighborhood. As the sinus groove is traced from below upward, it gradually, or in some cases rather suddenly, curves backward to join its parent, the lateral sinus. Between the upper surface of the bend of the sinus groove and the mastoid roof is found in some temporal bones a triangular space from which it is necessary to remove the diploic or cellular tissue. The rather thorough removal of the pneumatic or diploic structures throughout the mastoid is made necessary by the frequent finding of pus in cells deeply placed and at a distance from the main source of infection.

VIII. *Obliteration of the Zygomatic Cells.*—Placed superficially—*i.e.*, near the outer cortex—and corresponding roughly in position to the posterior root of the zygoma, is a collection of diploic or pneumatic spaces, popularly known as the zygomatic cells. They are to some extent present in practically all bones of diploic or pneumatic type. In very dense bones—*i.e.*, those approaching the sclerotic type—they are frequently altogether absent.

So far as I am aware, Whiting was the first to call attention to the importance of including these cells among those which should be removed in every case of suppurative mastoiditis operated upon. They may be exposed by continuing the removal of the outer cortex forward over the spine of Henle and along the posterior root of the zygoma, and extending

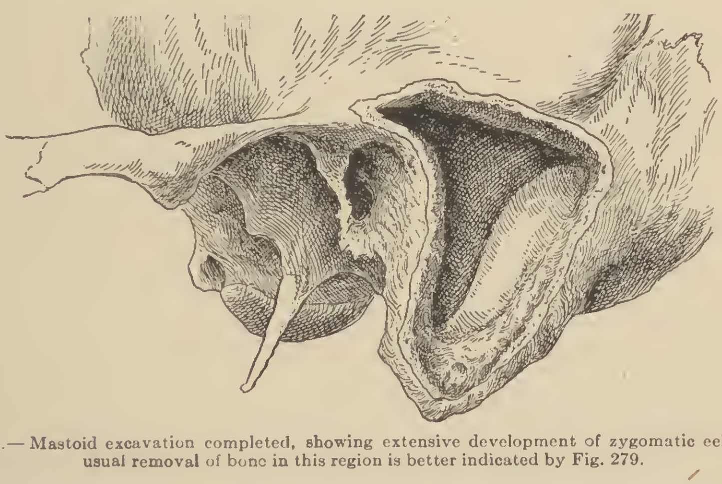


FIG. 231.— Mastoid excavation completed, showing extensive development of zygomatic cells. The usual removal of bone in this region is better indicated by Fig. 279.

this opening upward to the level of the inner plate which forms part of the floor of the mid-cranial fossa (Fig. 231). It is then an easy matter to obliterate and remove the cells thus exposed by means of a suitable curette.

Whiting claims that lack of thoroughness in removing these cells has been the cause of many operative failures.

Rational Thoroughness in Obliterating the Mastoid Cells.—Whatever changes in our conception of the mastoid operation have occurred within the past decade have been in the direction of greater thoroughness. We now leave no considerable collection of cells unopened in any part of the mastoid. The cells of the tip, at the base of the zygoma, in the depth of the anterior portion of the mastoid, and in the angle between the bend of the sinus and mastoid roof, and sometimes behind the sinus groove, are removed to the extent of outlining clearly these various parts of the general mastoid cavity. To Whiting belongs the credit of having focussed the attention of aural surgeons on the importance of systematic, routine thoroughness in obtaining these mechanical results. While there is no doubt that these changes have been in the right direction, it is a question whether our zeal or enthusiasm has not led us into certain errors of overstatement, which in turn have led, in some cases, to somewhat exaggerated views as to the mechanical results for which we should strive.

For example, if we say that every vestige of diploic or pneumatic cell structure should be carefully removed, we are advocating what in many bones is not only unnecessary but actually impossible. The accompanying figures (Nos. 232, 233, 234) were carefully drawn from a bone prepared

Fig. 232.—Line of section extending through mastoid process and petrous portion of the temporal bone.

by the writer. They show the presence of continuous chains of cells filling the mastoid, passing beneath the labyrinthine capsule and reaching to the apex of the petrous bone. The bone from which these illustrations were drawn is not unique. Even better than the pictures, a study of the bone itself impresses one with the futility of attempting to follow up and remove

literally all the cellular structures to which the mastoid operation may provide access.

There is one other point in regard to which the author wishes to record the results of his personal observations. A distinguished surgeon, in describing the technic of the mastoid operation, says, "The concluding step

Fig. 233.—Pneumatic spaces extending from mastoid process to apex of petrous bone.

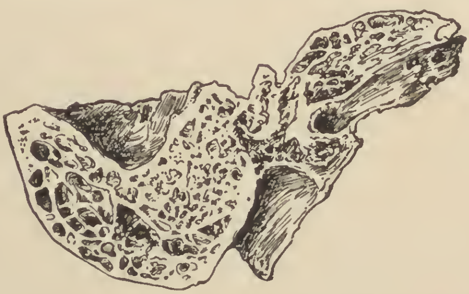


Fig. 234.—Continuous chains of pneumatic spaces extending from mastoid to petrous portion of the bone.

in the removal of bone now consists in smoothing off the rough diploic structure which invests the sigmoid groove *until the inner plate of the skull presents a glistening, even surface,* which sort of a polish a large dull curette moved gently and rapidly over it will speedily enforce." My own observation and experience have led me to believe that the above represents a super-refinement of technic which serves no particularly useful purpose and which may reduce to a dangerous degree the vitality of the bone. I have seen skilful operators so polish their bone surfaces as to create a bone cavity of striking smoothness, and in the same cases I have seen granulations form and local recovery occur only after a period of unusual delay. In my own operative experience I have repeatedly observed that when in the interest of thoroughness I have obtained a particularly smooth and

even surface, repair, as represented by the formation of a covering of healthy granulations, has been proportionately slow.

As aural surgeon to the Willard Parker Hospital for Infectious Diseases, I have during the past eight years had a fairly large experience in operating upon children suffering from infectious diseases. In these cases, it is certainly a fact that if we attempt by curettage to obtain smooth, even surfaces, we shall have failure of local nutrition and necrosis, calling for secondary operation, in an exceedingly large percentage of cases.

The point I wish to emphasize is that after we have eliminated all masses of cellular or diploic tissue which might be suspected of harboring concealed pus or foci of infection, and have thereby defined the limiting walls of the mastoid cavity, we have fulfilled the purpose of the operation. To go further and curette these walls into surfaces of even and polished smoothness may reduce their vitality to a point seriously interfering with normal repair.

Before taking up the post-operative treatment, a word should be said of certain operative incidents which occasionally occur, either by accident or intention, during the course of a mastoid operation.

Exposure of Dura.—It is not uncommon, either as a result of necrosis, accident, or the intentional removal of diseased bone, that the dura in contact with the cerebral surface of the tegmen antri is exposed. Unless the dura itself is punctured or torn, this occurrence need cause the surgeon no anxiety. An area of exposed dura presenting in the mastoid wound is certainly a safer condition than dura covered by doubtful or diseased bone. From the edges of the opening in the cranial plate any rough or sharp surfaces or points should be removed. The dura exposed through such an opening, if not itself lacerated, will usually be covered by healthy protective granulations even sooner than the adjacent bone surfaces.

Exposure of Sigmoid Sinus.—Accidental or intentional exposure of the sigmoid sinus, if its dural covering is not injured, usually leads to no untoward consequences. When a very small surface of the sinus has been accidentally exposed, it is safer, in the writer's opinion, to enlarge the opening to the extent necessary to expose an area at least three-eighths of an inch wide by a half inch long. This enables the surgeon to determine definitely that no traumatism of the dura has occurred, and in case of subsequent sinus trouble provides an opening which may be quickly enlarged to the extent required for further surgical intervention.

The surgical technic of exposing the sinus will be given in connection with the surgical treatment of infective sinus thrombosis.

Accidental Opening of the Sinus.—An experience which probably comes to every aural surgeon is that of accidentally nicking or tearing the sinus. The immediate result is naturally a very copious hemorrhage. Blood pours into the wound in a volume naturally disconcerting to the inexperienced surgeon. When the bone covering the sinus is diseased and adhesions between the sinus and inner bone surface have occurred, injury or tearing of the sinus wall may be difficult to avoid. It is a wise precau-

tion, therefore, to have in readiness a supply of small, tightly-wound rolls of sterile iodoform gauze, of the thickness and length shown in Fig. 235. One of these, taken up on the points of an ordinary thumb forceps, is usually easily applied over the bleeding point and will control the hemorrhage. If the hemorrhage is not controlled by one roll, another placed alongside of it usually will. The gauze roll is held by an assistant *upon the opening in the sinus, but on no account should be forced into it,* nor is it usually necessary to compress the vessel so as to obliterate its lumen or reduce its calibre. Bleeding having been controlled, the assistant holds the gauze roll in position, while the surgeon proceeds calmly and systematically with his operation.

Fig. 235. — Iodoform gauze plugs for control of hemorrhage in case of accidental opening of sinus.

The Element of Time in Mastoid Surgery.—In operating upon the mastoid the primary aim of a good surgeon is not speed, but the safety of the patient and completion of an adequate and perfect operation. On the other hand, prolonged anæsthesia is not without risks, and any unnecessary delay is an element of danger. Ordinarily a simple mastoid operation should be completed in a half to three-quarters of an hour, and only in a case of exceptional difficulty should it exceed an hour. The skilful surgeon works without apparent haste, but attains his result quickly because each thing he does accomplishes some essential step in the operation.

This deliberate but effective time-saving can be attained only by the adoption and rigid following of some systematic plan of attack plus the development of a certain degree of operative skill. If the surgeon begins to remove the tip, leaves it to define the sinus groove, and before this is completed is diverted to an attack upon the zygomatic cells,—in short, if he proceeds with his work without system and in haphazard order,—he may eventually complete a creditable operation; but progress is likely to be slow, and the chances that some essential step will be overlooked are increased. In describing the bone operation, I have therefore taken up separately the successive steps in the order which should be observed by the surgeon.

Postoperative Treatment.—Whatever changes have occurred in recent years in the theory and practice of mastoid surgery have related to the treatment of the soft parts rather than to the bone operation. The gauze-packed open wound has given place to the partially closed wound. This transition, though gradual and in no way spectacular, is of greater importance than is generally realized. To the patient it means a quicker average recovery, infinitely less suffering and a better average cosmetic result. It may be worth while, therefore, briefly to compare the two methods.

Open Wound Method.—By the older method, the wound cavity on com-

pletion of the operation was firmly packed with plain or iodoform gauze. The gauze was used in sufficient quantity to project somewhat above the level of the bone cavity, the flaps being thus held apart. Subsequent dressing: while the outer dressing was commonly changed within twenty-four hours, the wound packing was usually allowed to remain until the fourth or fifth day after the operation. When first removed it was not uncommon to find a clean wound-surface of practically bare bone. This was considered a perfectly satisfactory condition. Subsequently the dressings were changed on alternate days, the successive dressings differing from the first only in the fact that rather less pressure was used.

Generally speaking, the ultimate results from this treatment were fairly good. As offsetting this fact, must be mentioned certain recurring mishaps which seem to have been more or less directly attributable to the method employed:

(1) In a fairly large percentage of cases ending in recovery, healing was unduly delayed, a lapse of 10 to 12 weeks between operation and final healing being by no means uncommon.

(2) In a proportion of cases certainly larger than are met with to-day, secondary operations for removal of diseased bone were necessary.

(3) In a considerable percentage of cases, healing, usually considerably delayed, occurred with the formation of a permanent and unsightly depression behind the ear. This unfortunate deformity is comparatively rare to-day.

We would now regard all or any of these mishaps as logical results of the post-operative technic; for it is clear that the continued gauze packing might (a) inhibit the formation of the healthy granulations and thus prevent or retard recovery; (b) interfere with the local blood supply, with consequent loss of vitality and osseous necrosis at certain points; and finally (c) by inhibition of normal tissue repair, prepare the way for the inevitable growth of integument from the flap margins into the wound cavity, this giving rise to a disfiguring depression behind the ear.

The general tendency during the past decade has been away from the old plan of wide open gauze-packed wounds and toward more or less closed wounds protected by some form of drainage. Under favorable surgical conditions,—*i.e.*, in the case of wounds lined with apparently healthy bone or in which comparatively small areas of healthy dura have been exposed,—the method employed by most progressive surgeons to-day is about as follows: the wound cavity is flushed with normal salt solution and carefully inspected to see that no bone particles are allowed to remain. A wick of plain sterile gauze, so infolded that no threads may be left when it is withdrawn, is placed in the depths of the wound with one end at the aditus, the other emerging at the lower end of the wound. Over this the flaps are approximated and united throughout from the upper extremity of the wound to within half an inch of its lower, or tip, end. This may be done by metal clips or, as the writer prefers, by interrupted silk-worm gut sutures. The sutures are carried preferably through the entire thickness of each flap

so as to approximate and unite not only the skin, but also the periosteal, edges. Such a wound presents somewhat the appearance presented by Fig. 236. A gauze wick fills the auditory canal, the whole being protected by the usual large dressing of sterile gauze. The bandage used by the writer is comfortable, does not slip, and has the appearance shown in Fig. 237

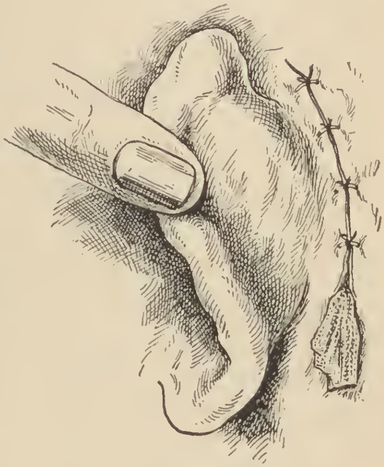


Fig. 236. Operation of mastoidectomy completed.

The dressings are changed daily. On the day following the operation the wick is withdrawn, and a pad or ball of sterile cotton is pressed gently against the lower wound-opening to take up by capillary traction any surplus serum or secretion. The wick removed is then replaced by a smaller one which is introduced only from three-fourths of an inch to an inch into the wound. Thereafter the wicks are changed at each daily dressing until there is evidence that they are no longer needed.

Dependence on the foregoing treatment is based on the hypothesis that the first wick used establishes a drain or pathway of least resistance along which pus or serum escapes as long as drainage is necessary. The smaller wicks used in subsequent dressings merely maintain the opening at the lower end of the suture line.

Fig. 237.—Mastoid bandage.

There are two fairly reliable signs that this treatment is progressing favorably, namely, (1) progressive and fairly rapid return of drum membrane and tympanic landmarks to a normal appearance; and (2) progressive diminution of discharge from the opening in the post-auricular wound.

I would abandon this treatment and open the wound in any case in which the tympanic structures did not within a reasonable time show progressive improvement. On the other hand, I might persist during a considerable period in spite of a fairly profuse, if diminishing, post-auricular discharge.

Interpretation of Pus.—It is almost a habit of mind to think of pus as a danger symptom since it always indicates a focus of infection, present or preexisting. In reality, pus *per se* represents danger only if confined or if its secretion, or production, is more rapid than its elimination. A raw or granulating wound without secretion is, of course, a surgical impossibility. Given, therefore, a partially closed wound from which we believe that we have eliminated the primary focus of infection, we need not be too ready to take alarm at a discharge which may represent only a stage in the process of repair.

I have known wounds treated by this method to have every appearance of being practically healed within ten days. More wounds are healed within three to five weeks. Some, for one reason or another, do not progress satisfactorily, but after separation of the flaps, there follows a more rapid process of healing than by the older method.

Theoretically this plan of treatment seems to meet the following requirements: (a) it provides reasonable and adequate drainage; (b) it utilizes every normal structure, bone and soft parts in the process of repair; (c) it secures the maximum vascular supply to the parts; and, perhaps most important of all, (d) it subjects the wound in process of repair to the least possible mechanical disturbance.

Contraindications.—That there are contraindications to this method goes without saying. As such I would consider surgical injuries to the sigmoid sinus, physical signs of disease in an exposed sinus wall, and unusual extension of bone necrosis.

Pronounced constitutional symptoms of septic absorption would also call for an open wound, since in such cases the systemic disorder, and not the local lesion, would be the first consideration.

In uncomplicated cases of suppurative mastoiditis the postoperative symptoms are usually not pronounced. During the first twenty-four hours, —sometimes prolonged to forty-eight hours—there may be moderate elevation of temperature, the so-called "postoperative temperature." In my experience, this is the exception rather than the rule. High temperature, and particularly continued high temperature, may indicate retention of secretion or pus, and would call for opening, and inspection of the depths, of the wound. After cleansing the wound of retained pus or infected clot, or correcting whatever condition may be at fault, the lips of the wound may be allowed to fall together, drainage being provided for as before by a small wick.

What has been said above refers to the general run of cases. Obviously, there are certain cases in which by reason of one or other of several conditions—*e.g.*, surgical accidents to dura or sinus wall, superficial changes in exposed dura, unusually wide-spread bone disease, etc.—safety may call for an open wound. In such cases, we now use light packing with sterile gauze until the bone cavity is lined throughout with healthy granulations, when the question of hastening recovery by a plastic closure operation may be taken into consideration.

Exuberant Granulations Springing from Carious Bone.—If areas of diseased bone are overlooked at the time of the operation, or if the vitality of

the bone is in certain regions destroyed, healing will not take place, and the need of opening the wound will soon be in evidence. The diseased areas are likely to be differentiated from surrounding healthy structures by one or other of two conditions, *i.e.*, either (a) the diseased bone is rough, bare and often devitalized in appearance; or (b) is covered with exuberant, flabby granulations. These granulations offer no resistance to the probe, which finds its way easily to the underlying rough surface which is characteristic of carious bone. When the diseased area is circumscribed and small, it may be possible under cocaine to remove with a curette the necrotic bone, after which rapid healing may take place. When, however, one has reason to believe that postoperative failure to heal is due to an extending area of necrosis, the shortest and safest road to recovery is usually by way of a secondary operation under full anæsthesia.

Arrested Repair.—A postoperative condition, of which I have seen no mention in text-books, but with which the surgeon has occasionally to deal, is characterized by the abortive type of granulations which line the bone cavity. Postoperative repair seems at first to follow a perfectly normal and favorable course. The bone cavity is soon lined by a layer of firm and apparently healthy granulations. The tympanic condition shows progressive improvement, and within a reasonably short period the drum membrane heals. In the postauricular wound, however, the process of repair is arrested. There is little or no pus, and in fact not more secretion than is inseparable from a granulating wound. The granulations themselves, though of healthy appearance, are stationary,—*i.e.*, the process of tissue-building seems arrested. Do what we may to increase the local blood supply and thereby stimulate a normal growth of new tissue, the aditus remains open, and the postauricular wound persists as a cavity of very considerable size. In my experience this condition—*i.e.*, arrested repair without evidences of osseous necrosis—is almost invariably an expression of anæmia or of some otherwise lowered constitutional state.

The treatment offers an alternative between two methods,—viz., (a) we may allow the skin from the edges of the wound to extend into, and line, the wound cavity, in which case we must prepare the patient for a permanently disfiguring depression behind the ear; or (b) we may close the wound by a plastic operation. In the writer's experience the latter has been a uniformly successful procedure. The operation requires complete anæsthesia and preparation of the operative field as for the original operation. The soft parts are elevated from the bone for a distance of an inch and a half, or more, from the posterior border of the wound. This results in a sort of pocket between the bone and its periosteal covering. The anterior flap, which has usually become strongly adherent to the subjacent bone, is lifted from its attachments. The object of these manœuvres is to release both the anterior and posterior flaps of the wound, so that they may be brought together without tension and sutured. If this requires forcible traction, the periosteum must be elevated from the bone to a greater distance behind the ear. This elevation of the posterior flap must

be extended until the edges of the wound can be brought together absolutely without tension. Unless this is done, the sutures will not hold,—*i.e.*, they will "slough out." Usually the skin has to some extent become infolded over the edges of the wound, and must be dissected up, or cut away, in order to bring raw surfaces together. The granulations within the mastoid wound are now removed with a curette and the cavity allowed to fill with blood. The prepared lips of the wound are now brought together and united by interrupted silkworm-gut sutures, except at the lower end of the wound. Here a minute gauze drain is introduced to a distance of not more than a half inch. A large protective dressing of sterile gauze is then applied. From the day of the operation, the dressings should be changed daily. On the day after the operation, removal of the wick from the lower end of the wound is followed by the escape of a considerable quantity of serum. Very gentle pressure over the suture line serves to further rid the wound of serum seeking escape. A wick similar to the one removed is then introduced. This is removed on the following day, after which, as a rule, no wick should be introduced. The sutures are usually removed on the fifth or sixth day, and a day or two later all dressings are discontinued.

In my experience, this procedure has been invariably successful in properly selected cases,—one indication of its suitability being a drum membrane and tympanum which have practically regained their normal condition.

As particularly typical examples of this class, I may cite two of my own cases,—one that of a chlorotic school-girl of sixteen, the other an old gentleman of sixty-seven years. Neither of these patients would have looked amiably upon the suggestion of a large permanent depression behind the ear. In both the blood examination showed a markedly reduced percentage of hæmoglobin. In each case the plastic operation was undertaken only after months of effort to stimulate sluggish, but otherwise healthy, granulations to more vigorous growth. The operation in each case was followed by healing within a week, without any disfiguring depression. Recovery has been permanent in each case.

Before leaving the subject of the mastoid operation, I wish to say a word as to the treatment of the tympanum both at the time of, and following, the operation. If the opening in the drum-head is not sufficient to provide free drainage, it is safer to reincise it at the time of the operation. At each change of dressings, the ear should be thoroughly cleansed of pus, and dried, and the canal packed with sterile gauze. Rapid improvement in the condition of the middle ear, with early healing of the drum membrane, usually foretells a fairly rapid healing of the mastoid wound. Lack of care in keeping the canal free of pus is probably a cause of failure, and certainly of delayed healing, in a certain percentage of cases.

The "Blood-clot Operation" (Blake, Reik).—The use of the blood-clot to promote early filling of the so-called dead space of the mastoid

wound, and incidentally to obtain primary healing of the outer wound, has added an interesting and instructive chapter to our knowledge of mastoid surgery. Since the bone operation does not differ from that in which the open method of after-treatment is used, the whole procedure may be described rather briefly.

The preparation of the patient—*i.e.*, operative field—is exactly as described for the older method. The drum membrane, even though showing a fairly large perforation, is reincised in order to provide the freest possible drainage from the tympanum. The postauricular incision through the soft parts differs from that used in the regular operation in being placed a little further behind the postauricular attachment,—*i.e.*, it commences at or below the tip and is carried upward following a curvilinear direction parallel with, but a half inch behind, the attachment of the auricle. Especial care should be taken not to injure the periosteum. Unless absolutely called for, the supplementary horizontal incision should be omitted, and, as the primary incision is a quarter of an inch further back,—*i.e.*, from the auricular attachment,—this additional space is available for the bone operation.

When the bone operation is completed, the cavity is washed out with normal salt solution, dried, and then allowed to fill with blood. The anterior and posterior flaps (soft parts) are then brought together, the edges carefully approximated, and held by interrupted sutures of silkworm gut. In this way the wound is entirely closed except at the lower end, which is left open. "The lower portion should be left free for serous outflow, and this outflow should be assured at each subsequent dressing" (Blake). Over this a large protective dressing of sterile gauze is applied.

The postoperative treatment consists chiefly of daily changes of the protective dressing, strict care being observed to do nothing which might carry infection into the wound. The stitches are removed in from five to seven days. In favorable cases the wound, so far as further care is required, may have every appearance of being quite healed in less than two weeks from the time of operation.

The facts which its advocates urge in favor of the adoption of this operation may be stated as follows:

1. The blood-clot itself has been proved to possess a decided bactericidal property; and this is depended upon to counteract any germs left in the mastoid wound, and to prevent reinfection.

2. In surgical diseases and injuries of the long bones the use of the blood-clot has provided a reticulum or scaffolding upon which the new tissues have seized,—the clot becoming quickly organized and finally converted into permanent osseous tissue. The cavity resulting from the mastoid operation is regarded as providing a favorable field for this conservative process.

3. In actual experience a large proportion of the cases operated upon by this method are said to have been successful.

4. When the blood-clot method is successful, the patient is saved much

postoperative pain and discomfort, and, in the case of wage-earners, the loss of time from work is only a small fraction of the postoperative period by the open method.

As to the danger of the closed wound in cases in which postoperative infection occurs, Reik holds that a remaining focus of infection will involve the clot itself sooner than normal living tissues, and that "the natural tendency, when a sufficient amount of septic material has been left in the wound to interfere with natural healing, is toward destruction of the clot and consequent breaking down of the wound margins; hence the dangers to life are purely hypothetical."

As to the cases in which the clot becomes infected, Blake says, "The persistence of the blood-clot during the period of its protective viability only, even though it then breaks down and comes away entirely, results in the formation of foundation granulomata, which are a basis for subsequent repair, with speedier and more satisfactory results in healing than are obtainable when the wound is dry-packed from the beginning."

In the writer's view, the chief objection to the adoption of the blood-clot method is that in cases of severe tympanic infection we are obliged to assume a postoperative escape of pus from the vault backward through the aditus into the space formerly constituting the antrum. Even if this does not result in postoperative reinfection of the entire wound, we are deprived of the use of the aditus as a means of draining the vault. In operating by the open method I believe that the usually rapid healing of the tympanic lesion is due very largely to postoperative drainage through the aditus. The blood-clot operation sacrifices this advantage. I should, therefore, expect tympanic resolution to be slower and less certain when the wound is closed at the time of operation than when the open method is employed. Those interested in the blood-clot operation are referred to the papers mentioned below.[4]

SURGICAL TREATMENT OF CHRONIC MIDDLE-EAR SUPPURATION.

Radical Mastoid Operation; Schwartze-Stacke Operation. — Definition: To otologists everywhere the term "*radical operation*" signifies an operation which aims to relieve chronic middle-ear suppuration by the conversion of the middle ear, antrum, and any diseased spaces within the mastoid into a single cavity, which is primarily drained through the external auditory meatus, and ultimately lined by integument continuous with that of the membranous canal. With complete epidermization, all mucopurulent discharge ceases.

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[4] Blake, C. J.: The Value of the Blood-clot as a Primary Dressing in Mastoid Operations, read at the Toronto meeting of the British Medical Association, 1906.

Reik, H. O.: The Blood-clot Dressing in Mastoidectomy, Considered Physiologically, Jour. American Medical Association, March 31, 1906.

Reik, H. O.: Some Facts and Figures relating to the Blood-clot Dressing in Bone Surgery, Trans. American Otological Society, 1906.

Sprague, F. B.: The Blood-clot Method of Wound Repair in Aural Surgery, The Laryngoscope, St. Louis, September, 1906.

The indications for the radical operation have already been stated (pages 216 to 217) and need not be repeated here. The writer wishes, however, to reiterate the great importance not only of making a thorough physical examination of the ear, but also of testing carefully the patient's hearing power, and also the functional soundness of the static or vestibular apparatus. The demonstration of fairly acute hearing power should certainly be weighed carefully in considering the advisability of an operation which sometimes influences the hearing unfavorably; and the evidence of a diffuse suppurative lesion of the labyrinth, even though all active symptoms may have long since subsided, is recognized as a contra-indication to the radical operation, unless we are justified in operating at the same time upon the infected labyrinth.

Preparation of the Patient.—The method of preparing the operative field has already been described. After the side of the head has been shaved, the auditory canal should be wiped out, then filled with peroxide of hydrogen, and finally irrigated with a 1 in 2000 solution of bichloride of mercury. It is then packed rather firmly with a strip of plain sterile gauze. The cleansing and sterilization of the auricle and the shaved area behind it are carried out by exactly the same method as for the simpler mastoid operation.

The incision begins below at the tip and follows a curvilinear direction behind the ear to a point above the upper attachment of the auricle, its centre being five-eighths of an inch behind the postauricular attachment (Fig. 238). From the tip to the temporal ridge this incision should divide all tissues down to the bone, while above the temporal ridge it may reach only the temporal fascia,—*i.e.*, it is not necessary that it should include the temporal muscle. In elevating the anterior flap thus formed, great care should be taken not to tear or mutilate the periosteum which, it must be remembered, is destined to form the outer wall of the cavity proposed by the operation. Richards has called attention to the following advantages of a very wide anterior flap,—viz., (a) when the posterior wound is closed, the suture line will lie behind the posterior margin of the excavation and have, therefore, the firm support of the mastoid cortex; (b) the outer wall of the bone cavity will be formed by an unbroken surface of periosteum, whereas, with a primary incision close to the auricular attachment, the suture line would form part

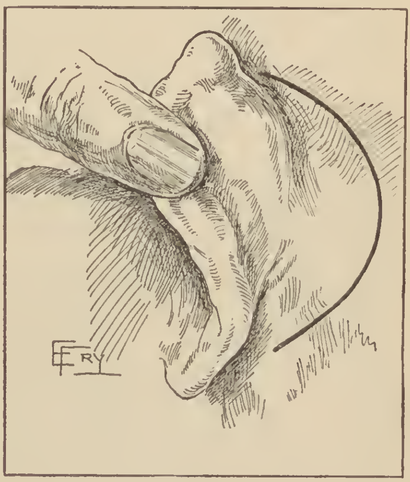


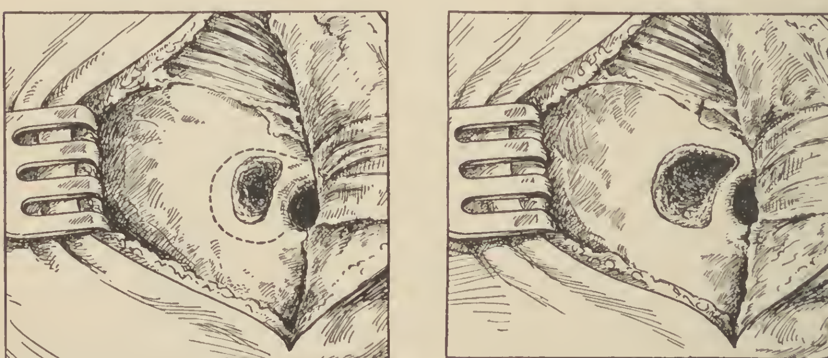
Fig. 238.—Postauricular incision for the radical operation.

of the outer wall of the bone cavity and granulations forming there might project into it and interfere materially with final healing.

The anterior flap is elevated and pushed forward so as to expose a large part of the outer rim of the bony meatus,—*i.e.*, posterior, superior, and inferior aspects. The membrano-cartilaginous meatus is at the same time completely separated from the posterior wall of the bony meatus by means of a narrow, curved periosteal elevator. The suggestion of Stacke, that the inner end of the membranous canal wall—*i.e.*, near the attachment of the drum membrane—be cut through with an angular knife, is quite unnecessary, since the membranous canal tears away easily at this point and with less traumatism than would be caused by the knife.

When the radical operation is not successful, its failure is usually a result either of postoperative neglect or of the surgeon's neglect or omission of some apparently minor technical point during the operation. In describing these essential details, we shall take up separately the successive steps of the operation in the order in which they occur in the actual operation.

I. *Removal of the Outer Wall of the Antrum* (Fig. 239).—Generally speaking, it is safer—and certainly for the beginner—to open the antrum before attempting to remove the posterior wall of the bony meatus. For this purpose, the gouge (Fig. 214) is the safest as well as the most convenient instrument. The initial removal of cortex is indicated by the excavation in Fig. 239. The gouge is made to enter the bone just above and behind the suprameatal spine, and, being carefully guided along the



FIGS. 239 and 240.—First stages of radical operation.

anterior border of the mastoid, removes a layer of bone not more than an eighth of an inch in thickness. Usually in cases of long-standing middle-ear suppuration, the cortex is very thick, and it is necessary to remove several layers of bone before the interior of the mastoid is reached. By hugging the posterior canal wall, and very gradually deepening the narrow

excavation thus outlined, we minimize the danger of injury to the sigmoid sinus. If, in removing the cortex, part of the outer margin of the posterior canal wall is shaved off, this does no harm, and simply anticipates the next step of the operation. As soon as the exposure of diploic tissue or pneumatic spaces shows that we have entered the mastoid, we should deepen this space by means of a curette, keeping its cutting edge at first directed somewhat forward and curetting in the direction of the posterior canal wall rather than backward. Using the instruments in this way, we are not likely to injure the sigmoid sinus, even should its course lie far forward in the anterior half of the mastoid. When we have removed the cellular tissue corresponding to the first opening in the cortex, it is well to explore the boundaries of this excavation by means of a silver probe, the end of which has been bent almost at right angles with the shaft. By this means we may obtain the following data,—viz.: (a) If its bent extremity finds a passage or opening leading from the upper end of the excavation forward into the tympanic vault, we know that we have entered the antrum; (b) by passing it directly upward, we ascertain the height or level of the tegmen antri; and (c) by passing it backward beneath the cortex, should a space here exist, we may determine the extent of overhanging cortex to be removed in this direction. It may be that this use of the probe will show the excavation to extend beneath the cortex to the extent shown by the dotted lines in Fig. 239. The cortex must then be removed up to the limits so outlined. This additional removal of bone completes the first stage of the operation, and leaves a bone excavation somewhat resembling that shown by Fig. 240.

Fig. 241.—Jansen's chisel.

II. *Removal of the Posterior Canal Wall.*—The outer two-thirds of the posterior canal wall, having no important surgical relations, may be removed rather rapidly. At the start, we may save time and prepare the way for the chisel by biting out a wedge-shaped segment with a rongeur. In using the rongeur we should remove the bone as nearly as possible along its attachment to the roof. After removing the outer half in this way, the chisel is by far the safer instrument. The remaining portion is now dealt with in the following way: With a Jansen chisel (Fig. 241), the rough upper margin left by the rongeur is chiselled away from the roof in thin vertical layers to the depth removed by the rongeur. The chisel is now used to lower the canal wall from without inward,—beginning below and cutting in thin layers upward and inward toward the roof. By using the chisel alternately along its outer margin and along the line of its attachment to the roof, the posterosuperior canal

wall is soon reduced to the narrow inner rim, or bridge, shown in Fig. 242. By this use of the chisel,—*i.e.*, along its attachment to the roof,—the thinnest portion of the resulting inner margin, or "bridge" (*a*), is placed high in the tympanum,—*i.e.*, above the level of the horizontal portion of the facial canal. Up to this stage of the operation the lower half of the posterior canal wall (*b*) has been lowered only in moderate degree, and certainly not sufficiently to endanger the descending portion of the facial canal which lies beneath.

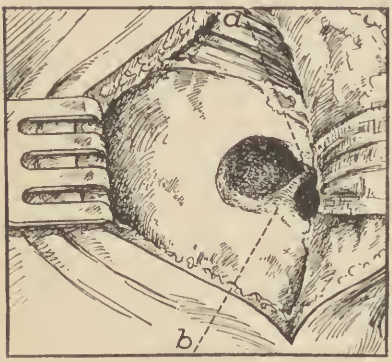


FIG. 242.—Radical operation; *a*, shallow "bridge" formed by inner margin of posterosuperior canal wall; *b*, remaining lower half of posterior canal wall.

The removal of the remaining rim of the posterosuperior canal wall presents some difficulty to the beginner on account of the danger of injury to the bend of the facial canal and horizontal semicircular canal which lie beneath. While the experienced aural surgeon has little difficulty in dividing the bridge with a chisel held firmly against its highest point, it is an instrument which must be handled with care. Körner uses a slender-bladed rongeur which removes the upper end of the bridge by a single bite. Richards believes that a small curette, introduced beneath the bridge from behind and pressed firmly against its upper attachment, is the safest instrument for this stage of the operation. It is a safe method only when the inner segment has been previously reduced to an extremely thin rim of bone. The writer some years ago invented a special forceps (Fig. 250), in connection with which he described a method of removing the posterior canal wall which eliminates completely the danger of injury to the facial nerve or horizontal semicircular canal. This method will be referred to later in connection with anatomical conditions for which it may be of special value. With a larger experience, the writer is inclined to believe that with the average run of temporal bones the work can be done quite safely by the careful use of either rongeur, chisel, or curette, according to the skill of the individual in the use of one or other of these instruments.

The method usually employed by the writer is as follows: The remaining inner margin of the posterosuperior canal wall having been reduced to a delicate bridge of bone having only a slender attachment above to the roof, a small straight-edged chisel is applied at this point —*i.e.*, hugging the roof—and the bone divided by a very slight tap of the mallet. The slightest tap is all that is required, the horizontal semicircular canal being endangered by a too vigorous blow. The bone having been thus divided above, a small curette is introduced from behind beneath the bridge, which is then lifted from its lower attachment, should

this not have been completely fractured. Removal of the bridge completes the second stage of the operation, and leaves a cavity similar to that shown by Fig. 243.

III. *Removal of the Roof of the Meatus.*—The posterosuperior canal wall having been removed, we should turn our attention to the plate of bone forming the roof of the meatus (Fig. 243). This should be so thoroughly removed as to bring the roof of the vault and the superior wall of the bony meatus into a continuously even or unbroken surface. This may be done with a sharp bone curette,—either the Richards instrument or the ordinary sharp spoon curette,—which removes the bone quickly from within outward. This part of the operation is of considerable importance, since a ridge of bone separating the roof of the attic from that of the bony canal provides an unfavorable surface for epidermization, and leaves an angular space at the junction of the roof and outer wall of the vault in which persistent granulations are likely to form.

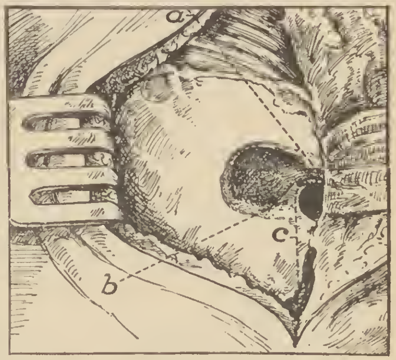


FIG. 243.—Radical operation, after removal of "bridge" formed by posterosuperior canal wall; *a*, roof of bony meatus; *b*, facial spur, formed by remaining lower half of posterior canal wall; *c*, dotted line indicating course of facial canal.

IV. *Preliminary Curettage of the Tympanum.*—If the malleus and incus have escaped destruction, it may be an easy matter at this stage of the operation to remove them with a tympanic forceps. In many cases, however, they have undergone complete or partial disintegration. Again, the necrotic remnants of the two larger ossicles may be hidden from view beneath masses of exuberant granulations practically filling the atrium and hypotympanic space. In removing such obstructive masses some care is necessary to insure against two particularly serious accidents,—viz., (a) injury to the horizontal portion of the facial canal, and (b) rupture or tearing of the capsular ligament which holds the stapes within the oval window. The latter accident has been the cause of more than one fatal case of purulent leptomeningitis. The curette having been introduced above and behind the obstructing mass, its edge should not touch or engage the inner tympanic wall,—the granulations being crowded downward against the floor of the hypotympanic space and thence swept out into the canal. We may then remove more thoroughly the remaining granulations,—*i.e.*, from the floor of the hypotympanic space, the region of the Eustachian orifice, etc.,—particular caution being observed, however, in using the curette in the neighborhood of the facial canal and niche of the oval window.

V. *Reduction of Ridge Representing Lower Half of Posterior Canal Wall.*—The next step is the removal of the bony spur left by the lower half of

the posterior canal wall. This is sometimes spoken of as "the facial spur," a term which for convenience of description shall be employed here. Our guide to surgical safety in this region is the bend of the facial canal (Fig. 243). Running more or less horizontally across the inner tympanic wall, this canal is indicated by a linear ridge which passes backward above the oval window, behind which it disappears beneath the upper extremity of the facial spur. Beneath the apex of this spur the facial canal bends downward, and thence descends almost vertically to its termination in the stylomastoid foramen. The facial spur may therefore be safely lowered

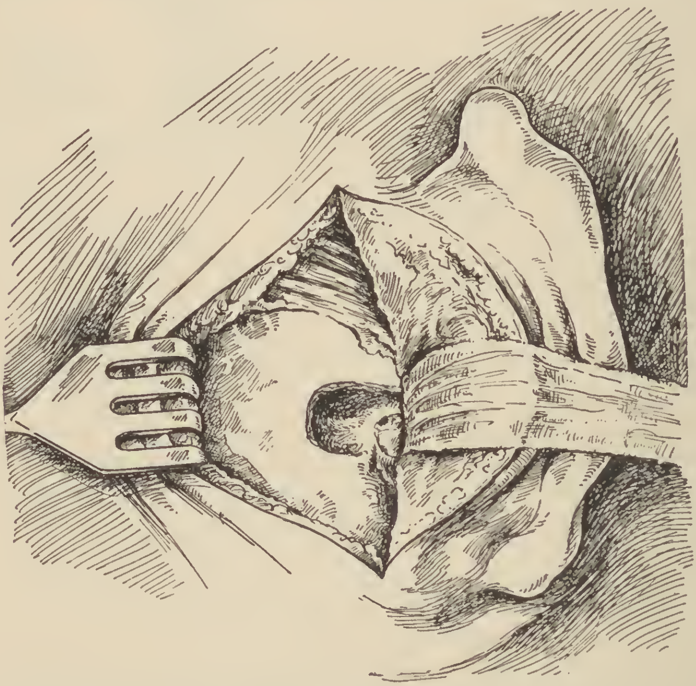


Fig. 244.—Radical operation, after lower half of posterior canal wall has been reduced so as to appear as a direct continuation of the facial ridge.

above until it has the appearance of being a continuation of the ridge representing the horizontal portion of the facial canal. The bend of the facial canal is also our chief surgical guide as to the course of the descending portion of the facial nerve, for below the bend of the canal its direction is almost vertically downward. The facial nerve lies, therefore, rather superficially beneath the upper extremity of the facial spur, but is very deeply situated beneath its lower part.

The actual work of reducing the lower half of the posterior canal wall may be done as follows: With a straight-edged chisel the bone is removed in successive layers from below upward. The first one or two layers of

bone removed may be rather thick below, where we know the nerve to be deeply placed beneath our point of attack. After this, however, we should keep constantly in mind the direction and course of the facial canal, making our removal of bone only in very thin layers, and as nearly as possible parallel with the course of the nerve. That is to say, if the chisel is made to cut in a direction at right angles to the course of the canal, the nerve may be completely severed, with consequent permanent paralysis. When the ridge has been reduced approximately to the dimensions we wish it to assume, it is safer to lay aside mallet and chisel, and complete the work with a bone curette. With a curette of fairly large size, we may remove any rough surfaces and at the same time lower the ridge gradually to the desired extent. While this work is in progress the anæsthetist should constantly watch the face and immediately notify the surgeon of any facial twitching which may indicate that the nerve has been mechanically irritated. When this work is complete, the bone cavity should have somewhat the appearance of that shown in Fig. 244.

Failure to deal adequately with the facial spur leaves a vertical prominence, or ridge, which more or less bars the way to the posterior end of the bone cavity, leaving there a space not easily reached or cared for during the period of postoperative treatment.

VI. *Removal of Outer Wall of Hypotympanic Space.*—In Fig. 245 the hard, dense bone of the floor of the meatus is shown in striking contrast with the rough depression constituting the floor of the tympanum. The hypotympanic space frequently contains a considerable amount of diploic tissue. This diploic tissue must be scraped out and the inner margin of the floor of the meatus removed with a curette. The floor of the tympanum and contiguous portion of the floor of the canal must then be scraped to a smooth or unbroken surface. When this part of the operation is neglected, the hypotympanic floor frequently becomes the seat of persistent granulations which prevent healing and resist all therapeutic measures short of a secondary operation.

Fig. 245.—Tympanic floor.

VII. *Curettement of the Eustachian Orifice.*—The next step of the operation is the very thorough curetting of the Eustachian orifice. Here almost invariably in chronic middle-ear suppuration is found a diseased mucosa. Very frequently the tube is blocked by exuberant granulations and it may be the seat of osseous necrosis. The treatment is a careful and thorough curettement. The anterior wall of the tympanum merges

Fig. 246.— Ring curette.

centrally into the funnel-shaped mouth of the bony tube. Into this a small curette may be introduced for some distance,—*i.e.*, 5 or 6 mm. The small ring curette shown in Fig. 246 is provided with sharp edges and is well adapted for this work. After removing any masses of granulation tissue, the various walls of the tube are gently scraped until the curette encounters smooth bare bone in all directions. Our purpose is not only to rid the tube of obstructing granulations, but also to remove completely its mucous lining. The logic of this treatment becomes clear when we remember that obliteration of the lumen of the canal is exactly what we wish to attain. When we have thoroughly removed the mucous lining and curetted the bony walls into even surfaces, the production of healthy granulations would seem inevitable, and granulating surfaces in so narrow a tube should lead logically to firm cicatricial stenosis.

The writer is inclined to believe that the frequently reported failures of the radical operation to relieve the discharge from the Eustachian tube are to be explained almost wholly as due to a lack of thoroughness in carrying out the work above outlined.

Just below and internal to the Eustachian canal—*i.e.*, separated only by its lower internal wall—is the canal for the internal carotid artery. While injury of the carotid artery is, therefore, theoretically possible, and while such accidents have been reported, I have personally known of no such cases, and believe that with reasonable care and gentleness in the use of the curette it is not likely to occur. Just above the Eustachian canal is the incomplete canal for the tensor tympani muscle. Removal of the plate forming the floor of this canal, and incidentally of the tensor tympani muscle itself, is followed by no untoward results and actually leaves a more favorable condition for prompt healing at this point.

In our final survey of the work accomplished, it is necessary to recognize the following structures: Passing along the floor to the anterior tympanic wall one encounters first the Eustachian orifice (Fig. 247, *a*), immediately above which is the floor-plate of the tensor tympani canal (*b*). Above this, and separated from it only by a very limited space, is the beginning of the horizontal portion of the facial canal (*c*). This is undoubtedly the region in which injury to the facial nerve most frequently occurs. From this point the facial canal is traced as a linear ridge passing horizontally backward across the tympanum and disappearing beneath the vertical ridge representing the

remains of the posterior canal wall (*f*). Beneath the posterior end of the horizontal facial ridge—*i.e.*, just before it bends to pass downward beneath the posterior canal wall—is the niche of the oval window (*d*). Between the horizontal facial ridge and the hypotympanic space is the promontory (*h*), behind which is the niche of the round window (*g*). Just above the posterior half of the facial ridge is the horizontal elevation of dense, ivory-like bone, representing the anterior end of the horizontal semicircular canal (*e*). When the surgeon has sufficiently cleared the tympanum of its diseased mucosa and granulations, most of the above landmarks stand clearly revealed. I say "most" of them, because I wish to note an occasional exception in the case of the oval window. We must trace and outline the horizontal portion of the facial canal in order to avoid injuring it. We know the position of the oval

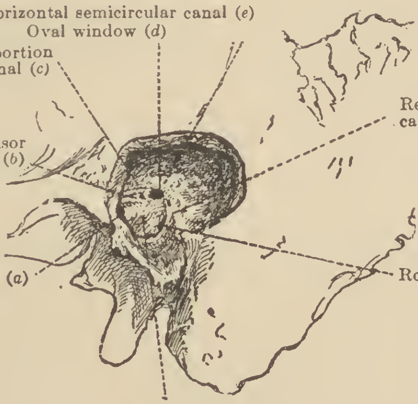


Fig. 247.—Tympanic structures usually exposed in radical operation.

window from its relation to the bend of the facial canal. In some cases the oval niche and even the stapes itself are in plain view; in others the stapes is covered and the niche filled by granulations. I do not agree with those who believe that granulations should be removed from the oval niche for the purpose of investigating the condition of the stapes and stapediovestibular joint. If we have made a thorough preliminary examination we shall have determined in advance the presence or absence of a labyrinthine fistula. A fistula being absent, granulations surrounding the stapes and within the oval niche may represent as distinctly a protective and conservative process as do granulations upon the dura covering the sigmoid sinus, and in neither situation should they be removed. The list of recorded cases of stapedial injuries leading to suppurative labyrinthitis has already reached an impressive number. The writer personally knows of two deaths directly traceable to stapedial injury during a radical operation. So far as he knows, granulations covering

the oval window do not usually indicate osseous necrosis, and do not interfere with postoperative healing. Furthermore, such granulations usually disappear spontaneously during the period of postoperative repair, leaving the depression of the oval window and sometimes the head of the stapes in plain view.

Facial Paralysis.—Before leaving the discussion of the bone operation, a word should be said as to the occasional accidental injury of the facial nerve. Much has been said of the danger of injuring the nerve during the removal or reduction of the posterior canal wall. But this danger—which with a careless or incompetent surgeon is quite real—is so obvious that most aural surgeons are forearmed against it. As a matter of fact, the nerve is not very frequently injured at this stage of the operation. Much more frequently the injury occurs while the surgeon is using the curette upon some structure within the atrium. The smallness of this operative field, the difficulties of keeping it free of blood, and the extreme thinness and brittleness of the bone forming the outer wall of this part of the facial canal combine to place the nerve here in an exceedingly vulnerable position. The surgeon can not, therefore, be too careful in using the curette within this space. If blood collects in the wound and obstructs his view, he may pack the wound with gauze saturated with adrenalin chloride, 1 in 1000. After this has remained in position one or two minutes, he will usually have the advantage of a fairly dry field. Before proceeding with his operation, he should take time to locate definitely the various tympanic landmarks, but particularly the horizontal portion of the facial canal.

Temporary facial paralysis occurs in certain cases in which the nerve has not been exposed at any point. In such cases the paralysis is usually of more or less gradual development,—that is, it is not complete when the patient recovers from the influence of the anæsthetic,—and must be explained as due to the pressure of inflammatory exudates upon the nerve or to a slight traumatic neuritis resulting from the use of instruments near the facial canal. The fact that a postoperative facial paralysis is not complete from the start is proof that the nerve has not been actually severed; some restoration of function may therefore be relied upon. In most cases the restoration is complete,—in others only partial.

When the face is found completely paralyzed at the moment when the patient regains consciousness, the condition may cause the surgeon more anxiety. If he has exposed the nerve extensively and recognized it, so that he is sure that it has not been actually severed, he may expect rather confidently that the facial control will ultimately be regained. If, however, he has completed his operation without any knowledge of having injured or exposed the nerve, and yet finds complete paralysis as the patient recovers from the anæsthetic, the condition is one which naturally causes greater anxiety, since, having injured the nerve unknowingly and to the extent of complete ablation of its function, he is left in doubt as to whether

it has actually been severed. In such cases, the anxiety of the surgeon can be relieved only by the degree of confidence he may feel that the operation as he has performed it could not possibly have produced actual division,—*i.e.*, loss of continuity.

Facial paralysis following the radical operation is temporary in the vast majority of cases. When complete—*i.e.*, presenting typical reactions of degeneration—months may elapse before any signs of functional improvement are discernible. It is a most distressing complication, and the surgeon can not be too careful to guard against its occurrence.

Having completed the various steps of the operation as above described, it is well to scrutinize carefully the entire bone cavity for small areas of diseased or suspicious bone which may have been overlooked. Any rough points or surfaces should be made smooth, and for this purpose a spoon curette passed rapidly and lightly over the bone is a most effective instrument. Overhanging margins of cortex should be removed and the resulting edges made smooth. It is of considerable importance that the completed operation should leave the Eustachian orifice in clear view during the period of postoperative treatment and repair. In certain bones it will be found that the central portion of the anterior wall of the bony meatus bulges so prominently into its lumen as to obstruct considerably one's view of the Eustachian orifice. This difficulty can usually be corrected by reducing the thickness of the anterior canal wall with a suitable curette (Richards). In the case of unusually narrow canals, the writer has frequently obtained very useful additional space by this method. It is a point of considerable importance.

Before describing the plastic work upon the membrano-cartilaginous canal by which the radical operation is completed, it will be necessary to refer very briefly to certain variations in the method of operating upon the bone.

Zaufal-Heine Operation.—Zaufal, Heine, and many aural surgeons of note prefer to open the antrum by the preliminary removal of the posterior wall and roof of the bony meatus. As Heine describes it, it is performed somewhat as follows: Using a small gouge, the bone is entered just behind the suprameatal spine and a shaving of bone from the posterosuperior canal wall removed. If a small enough gouge is used, the outer three-fourths of the canal wall may be removed. The gouge is then applied to the contiguous portion of the roof of the meatus, and a layer of equal length removed. By using the gouge alternately upon the superior and posterosuperior walls, the pneumatic or diploic structures behind them are exposed, and the antrum, if of average size, is opened. When by this process the outer four-fifths of the posterosuperior canal wall have been removed, there remains the slender "bridge" of bone formed by the inner margin of this wall of the bony canal. This may be removed by means of a small curette or chisel, as in the regular operation already described. When this has been accomplished, the operation reaches the

stage shown by Fig. 248. The outer mastoid cortex must now be removed to a variable extent above, behind, and below the suprameatal spine,—usually to the extent indicated by the dotted line in Fig. 248. From this point the operation is completed by exactly the same steps as have been described in the previous pages.

Fig. 248.—Stacke's operation.

The above method with slight modifications has been for some years in use by many American surgeons. The technic varies from that of the Zaufal–Heine operation chiefly in that the mastoid cortex and posterosuperior canal wall are removed simultaneously. A rather large gouge enters the bone behind the suprameatal spine and removes a layer from the cortex above and behind the canal and from the posterosuperior canal wall at the same time. Successive applications of the gouge remove shavings from the anterior edge of the cortex until what is expected to represent the posterior limit of the excavation is reached. In this way the removal of the cortex and of the canal wall proceed simultaneously. The removal of cortex progresses sufficiently rapidly to provide adequate space for the gradual and comparatively easy removal of the canal wall. Finally, when only a narrow inner margin, or "bridge," remains of the posterosuperior canal wall, the operation reaches practically the stage shown by Fig. 242, from which point the technic does not differ from that already described.

Stacke's Operation.—Stacke's operation proposes the exposure of the tympanic vault and the antrum by chiselling away first the inner extremity of the roof and posterosuperior wall of the bony meatus. It differs from the Zaufal-Heine operation in that the gouge, instead of being applied first to the outer margin of the canal wall and removing the same in layers from without inward, is applied first to the canal wall near its inner extremity and the bone removed in small portions from within outward. The steps of the operation may be described somewhat as follows: A postauricular incision parallel with the auricular attachment having been made, the anterior flap and auricle pushed forward, and the membrano-cartilaginous canal having been separated from the bony meatus and either drawn out of the canal or crowded well forward, the bone operation is carried out as follows: The malleus, if within reach, is removed by means of a tympanic forceps. The next step is the removal of the inner extremity of the roof of the meatus by means of a narrow gouge the end of which is slightly curved to facilitate its engaging the bone. With a view to protect-

ing from injury important tympanic structures,—chiefly the facial canal and horizontal semicircular canal,—Stacke's "protector," a narrow flat instrument the terminal end of which is bent to an angle of 45° (Fig. 249), is introduced into the meatus, the end being carried upward into the attic. This instrument is held in position by an assistant. The surgeon then introduces his gouge, and chisels away the roof of the meatus from within outward until the superior wall of the vault and that of the meatus lie in the same plane. The protector is then rotated so that its terminal bent portion passes backward into the aditus or antrum, and the contiguous portion of the posterior wall chiselled away from within outward. This, of course, provides a free opening into the antrum by way of the meatus. Finally the cortex is removed over the usual area.

Fig. 249.—Stacke's protector.

Stacke's operation, in the writer's opinion, is one of the most dangerous procedures proposed in aural surgery. The possibility of injuring the facial or semicircular canal, either by the gouge itself or by a splinter of bone from the inner margin of the canal, can not be doubted. The use of the protector hardly seems to remove this danger, for, with the protector in place, the small safety space between the inner and outer walls of the vault is sacrificed, and any slip of the gouge or miscalculated force in the mallet blow is communicated to the "protector" and by it transmitted to the bone structures beneath.

There is but one condition known to the writer in which the tympanic end of the posterosuperior canal seems the only safe point of attack for opening the antrum,—viz., when the sigmoid sinus literally overlies the antrum and is in actual contact, or very close proximity, with the posterior meatal wall. This condition, though exceedingly rare, does exist. The writer has met with at least one such case in his operative experience, and has in his collection a temporal bone in which the position of the sinus-groove is such as to render it a physical impossibility to have entered the antrum by any route having a starting point upon the outer mastoid cortex.

Since the necessity for opening the antrum from the tympanic end of the meatus occasionally confronts the surgeon, the writer wishes to refer

briefly to a method of attack which he described in a paper published in 1904,[5] and which has the advantage of absolute safety. The special instruments required are the cutting forceps shown in Fig. 250. Two sizes are needed. The following description is quoted from the paper in question:

Fig. 250.—Kerrison's tympanic rongeur.

Operation.—"The patient is prepared for operation and the mastoid cortex exposed in the usual way. The anterior flap and the auricle are pushed forward so as to expose the spine of Henle and entrance to the bony meatus, and the skin and periosteal lining separated from its posterior wall, roof, and floor from the entrance of the canal to its termination at the annulus tympanicus. The auricle is drawn forward by some form of retractor, preferably a strip of sterile gauze passed through the membranous canal.

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[5] Kerrison: A Bone Forceps for Use in Tympanic Surgery; Its Value in Safeguarding the Facial Nerve, etc., Laryngoscope, May, 1904.

"From this point it will be necessary to work by reflected light or by direct illumination by means of an electric forehead lamp.

"Before proceeding further, the fundus of the canal should be inspected. The remains of the drum membrane should be removed, and, if either malleus or incus be within view, an attempt should be made to remove it. If neither malleus nor incus be visible, the end of a small tympanic probe, bent near the tip so as to describe a right angle, should be introduced into the tympanum and carried upward into the vault. Should the tip of the probe encounter one of the ossicles blocking the entrance to the vault, it should be pushed backward or forward out of the way, and no attempt should now be made to remove it. We are now prepared for the first step of the bone operation.

"In using these forceps the writer has found it easier to remove first the roof and posterior wall of the meatus, the mastoid cortex covering the antrum being removed later.

"The small forceps (Fig. 250), with cutting edges separated and pointing upward, is introduced into the tympanum and carried upward into the vault. On attempting to withdraw it, the distal cutting edge will be engaged by the inner margin of the roof of the meatus. The handles are now gradually approximated, when the blades will be felt to crush or cut through the bone, and may be withdrawn, bringing away a small portion of the upper canal wall. Having cleared the instrument of bone, it is again introduced into the canal and made to engage the roof in the notch made by the first bite of the forceps. By a repetition of this process the roof of the meatus is quickly removed. The vault is now exposed to view and a bent probe may be passed backward into the antrum.

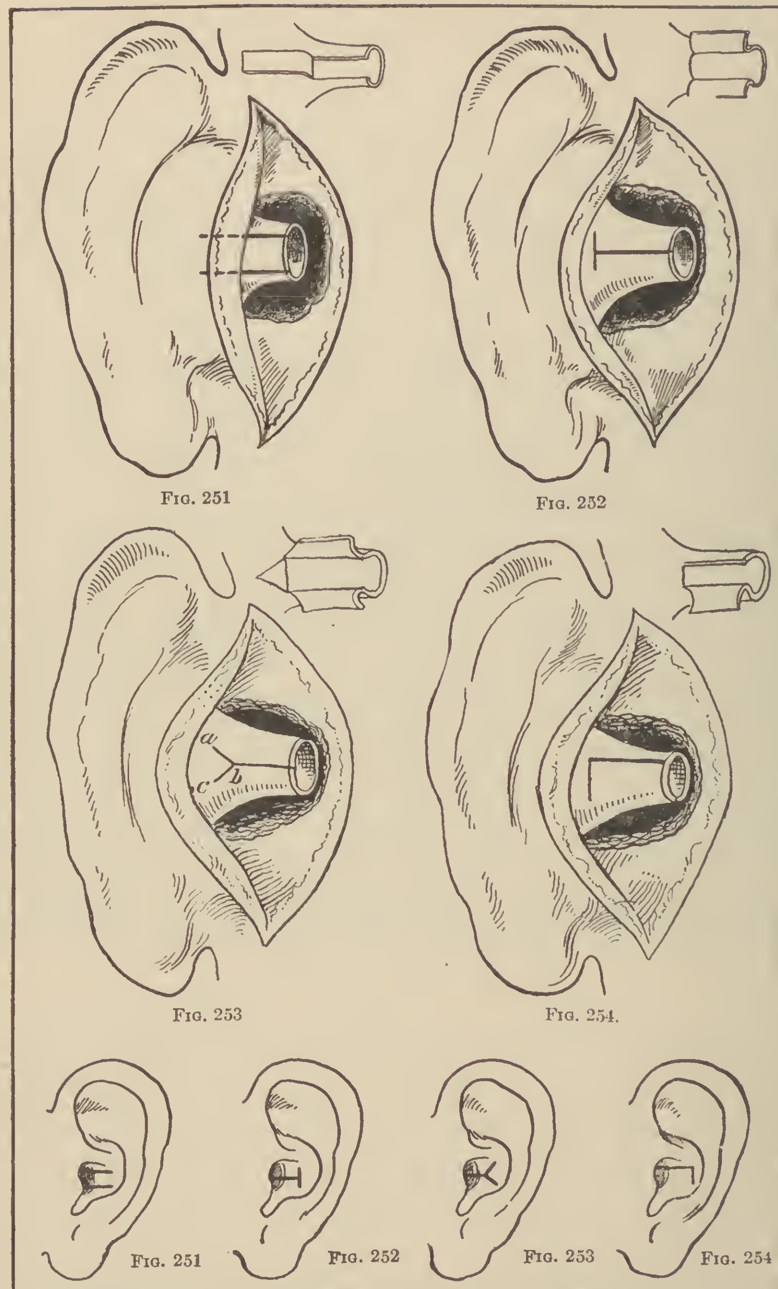
"The next step will be the removal of the posterior canal wall. For this purpose the small forceps, or the larger one if it will enter the tympanum, is employed. It is introduced into the canal as before described except that its cutting edges are now directed backward toward the antrum. The handles are carried slightly forward in order to bring the cutting edges in contact with the inner margin of the posterior canal wall, which is rapidly removed as far outward as the spine of Henle.

"The work just described—*i.e.*, the removal of the superior and posterior canal walls—can be done quite rapidly, usually requiring not more than five to eight minutes. The tympanic space and landmarks, including the oval window, horizontal portion of the facial ridge, and prominence of the external semicircular canal, are now well exposed to view, and are kept under observation during the remainder of the operation, which may be completed by the usual method."

This method of operating has seemed to possess the following advantages over Stacke's operation:

1. Considerable saving of time in removing the roof and posterior wall of the meatus and exposing the antrum.

2. Diminished danger to important structures, from the fact that the



Figs. 251, 252, 253, 254.—Diagrams illustrating various plastic operations forming part of the radical operation.

bone is cut in a direction from within outward,—*i.e.*, away from the stapes, facial canal, and horizontal semicircular canal.

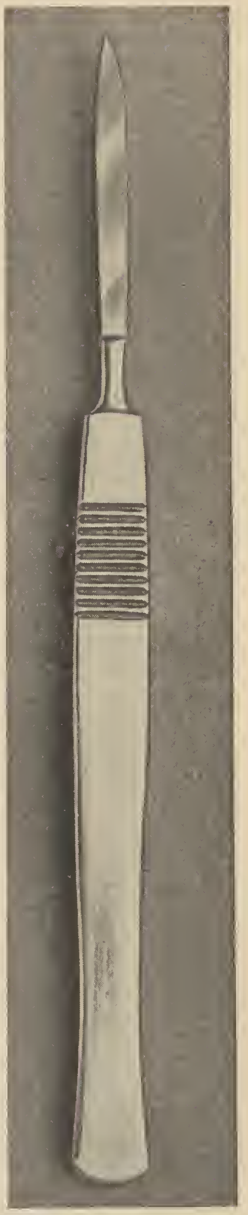


FIG. 255.—Narrow-bladed knife required in the plastic work of the radical operation.

The term "radical operation" no longer refers specifically to any particular method, or technic, and is used to describe any surgical procedure which converts the tympanic cavity and mastoid antrum into one large cavity of healthy bone, draining easily by way of the external auditory meatus. This mechanical result having been attained, the postauricular wound may be closed throughout. Before this can be attempted, however, it is necessary to adopt some form of plastic operation by which the membrano-cartilaginous canal can be made to conform to the enlarged bony space. A variety of excellent meatal flaps have been suggested, only four of which, however, need be described here,—viz.: (1) Körner's flap, (2) Panse's, (3) Siebenmann's, and (4) Ballance's.

THE KÖRNER FLAP (Fig. 251).—Körner's operation converts the posterior wall of the membrano-cartilaginous meatus into a tongue-shaped flap which is utilized as a partial lining for the posterior wall of the bone cavity. It is made as follows: A narrow-bladed knife (Fig. 255) is introduced into and through the membrano-cartilaginous meatus, with cutting edge directed backward, and the canal divided from within outward along the line of junction between roof and posterior wall. Within the concha it is carried backward through the skin and conchal cartilage following a line just below and parallel with the conchal end of the helix. As a rule, it should not extend backward further than midway between the anterior and posterior boundaries of the concha. The knife is then reintroduced and a similar incision parallel with the first, but hugging the floor of the membrano-cartilaginous canal, is made through canal and concha, ending at a point in the concha below the termination of the first incision (Fig. 251, small figure). If all structures of the membranous canal have been divided, the tongue-shaped flap between the two incisions may now be lifted out through the meatus. Catching the extremity of the flap with toothed forceps and drawing it backward, the surgeon uses its skin covering as the part to be preserved, and carefully dissects from it all subcutaneous structures, including the cartilage. This dissec-

tion is carried backward as far as the posterior extremities of the two parallel incisions. The meatal and conchal cartilage thus exposed is then cut away. This usually completes this part of the operation.

Prof. Körner himself no longer removes any cartilage from the concha. I have found this, however, a very useful procedure, as it enlarges the meatal opening, not enough to produce any noticeable deformity, but it does give valuable space through which to carry out postoperative treatment. Some operators, before closing the posterior wound, suture the meatal flap back against the anterior flap of the postauricular wound with a catgut suture. Personally the writer has not found this necessary. After cutting the flap, he has proceeded to close the postauricular wound, either by interrupted silkworm-gut sutures or by the metal clamp sutures. The concho-meatal flap is then carried to its place against the posterior wall of the bone cavity and held there by a firm packing of iodoform gauze.

The Panse Operation (Fig. 252).—This operation makes of the posterior wall of the membrano-cartilaginous canal two flaps,—a superior and an inferior. The narrow knife is introduced, as before, through the membranous canal, and a longitudinal incision made through the centre of its posterior wall. This incision includes the cartilage and is carried backward into the concha, usually to its mid-point (Fig. 252). The knife is now inserted at the posterior extremity of the first incision, and two vertical incisions—*i.e.*, one upward and one downward—are made at right angles to the first. This T-shaped incision provides for an upper and a lower flap. The skin is now dissected up from each flap, and the conchal and meatal cartilage so exposed is excised. When these flaps have been placed in position, and held there by catgut sutures or preferably by careful packing of the wound, they form a nucleus for the future epidermal lining of the roof, anterior wall, and floor of the bony cavity. The posterior wall is not provided for, but may be covered later by skin-grafts, or in successful cases is covered by the rapid spread of epidermis from other parts.

The Siebenmann Flap (Fig. 253) up to a certain point closely resembles that of Panse. The membrano-cartilaginous canal is divided by a longitudinal incision which passes through the centre of its posterior wall, but ends at the anterior margin of the conchal cartilage. From the posterior extremity of this incision, two short incisions extend, one upward and backward and the other downward and backward (Fig. 253). This provides for three flaps,—viz., a broad upper flap (a), a lower flap (b), and a short pointed or triangular flap (c). The skin covering each of these flaps must now be dissected up and the underlying cartilage removed. This method offers the advantage over Panse's operation that the short posterior flap provides a covering of skin for the posterior margin of the meatus, and a point from which epidermization may proceed over the posterior wall of the bone cavity.

Besides the above there are a number of excellent plastic methods, including that of Ballance, of London (Fig. 254), which is particularly suitable when it is desirable to secure considerable enlargement of the orifice of the membranous meatus. Any of these operations will give favorable results

in suitable cases. Another modification, which has come to the writer's knowledge since the accompanying illustrations were drawn, is the method introduced by Brühl. Brühl's operation is a modification of Panse's,—*i.e.*, the T-incision of Panse is supplemented by two parallel incisions passing backward through the concha and providing a short Körner-like flap. Brühl's operation is therefore a combination of the salient features of the Panse and Körner methods.

From the foregoing description it will be seen that in appropriate cases the completion of the bone operation is immediately followed by the plastic work upon the membranous canal and complete closure of the postauricular wound. There are, however, several conditions in the presence of which it is safer, in the writer's opinion, to postpone the closure of the wound. These may be mentioned in the following order: (a) Exposure plus injury—*i.e.*, tearing or puncture—of the dura at any point; (b) extensive exposure of the sigmoid sinus; (c) mid-cranial epidural abscess; perisinous abscess; and (d) large cholesteatomatous masses within the antrum and adjacent mastoid spaces. However thorough may have been our removal of cholesteatomata, it is safer to keep under inspection the surfaces from which they have been removed until these are completely covered by healthy granulations.

I am aware that many distinguished surgeons will not agree with me in regarding all the conditions above referred to as contra-indications to the immediate closing of the wound. Any of the above conditions, however, may lead to serious complicating lesions, and it seems to me more in accordance with surgical principles to keep the wound open until all doubtful areas are covered by a layer of healthy granulations.

When the bone operation results in a cavity whose walls in all directions are formed of healthy bone, the plastic work upon the canal and closure of the posterior wound should form part of the original operation.

Postoperative Treatment.—The postoperative treatment is almost or quite as important as the operation itself. That is to say, the surgeon who operates—no matter how carefully—and leaves the after-care of his cases to others will surely obtain but poor average results.

Ordinarily the first dressing should not be changed, or at least the gauze packing within the bone cavity should not be removed, until the third or fourth day after the operation. In some cases removal of the first dressing reveals a practically dry wound, in which cases the bone cavity should at once be repacked, this time with plain sterile gauze. If, on the other hand, the dressing is found saturated with serum or pus, the cavity should be carefully wiped dry with pledgets of sterile cotton, dusted with dry boric acid powder, and then firmly packed with strips of sterile gauze. After removal of the first packing, the dressing should be changed at least as often as on alternate days. In some cases it is advantageous, or even essential, to change the dressings daily.

It is important that at each change of dressing the wound should be made as nearly as possible dry before the gauze is replaced, and that the space representing the original tympanic cavity should be packed as tightly

as possible without exerting dangerous pressure upon the facial canal. The necessity for tight packing is clear when we remember that our purpose is to retard the growth of granulations beyond the thinnest possible layer covering the tympanic walls, and to encourage the rapid spread of epidermis from the flaps partially lining the bone cavity. Experience has taught that the process of epidermization progresses not only more surely, but also more rapidly, under a tight packing than in a lightly packed cavity. In a loosely packed bone cavity, granulations are likely to grow rapidly at certain points and effectively check the spread of the epidermis which otherwise would soon line its walls.

When, in spite of our efforts at prevention, the rapid growth of granulations in certain regions interferes with the process of epidermization, some means of removing them must be found. The use of strong solutions of nitrate of silver as a means of reducing redundant granulations has never in my hands given satisfactory results. When granulations must be removed, we shall save time and accomplish our work more effectively by means of a curette. The bone cavity should be packed with sterile gauze saturated with a 10 per cent. solution of cocaine. After this has been allowed to remain in position ten minutes, the offending granulations may be removed with a curette without discomfort to the patient, and usually without troublesome hemorrhage. The rapid reproduction of the granulations should be guarded against by very careful and firm packing.

There are certain cases—particularly those in which cholesteatomata have been present—in which during the first weeks following the operation the wound at each change of dressing is found to be bathed in a purulent secretion of very offensive odor. I have known such cases in which no physical evidences of remaining osseous necrosis could be found, and which ultimately went on to perfect healing. Peroxide of hydrogen is an efficient temporary deodorizer in such cases, but neither the peroxide, bichloride of mercury, nor wet dressings of permanganate of potassium prevent the return of the odor. I have found that dry boric acid powder properly used will correct this condition more quickly and with greater certainty than any other agent. It should not be introduced by a powder-blower, but carried into the wound in considerable quantity upon the blade of a spatula or upon any flat dull instrument. The bone cavity having been half filled with the powder, a strip of sterile gauze should be firmly packed against it. The action of dry boric acid powder used in this way is threefold,—*i.e.*, (a) it effectually deodorizes the wound, (b) it reduces the amount of pus secreted, and (c) it seems to exert an inhibitory influence upon the growth of new granulation tissue.

Certain cases which do not respond favorably to the measures above outlined will occasionally do well under the following treatment: The wound, having been wiped dry of fluid pus, is irrigated with a warm solution of boric acid and again dried. The bone cavity is then completely filled with dry boric acid powder, and the canal protected by a pad of sterile cotton placed in the concha. Each day the powder is removed by irrigation, the cavity carefully dried, and again filled with boric acid powder.

I have had under my care cases which healed perfectly under this treatment, which I had believed would eventually require a secondary operation.

Use of Dakin's Fluid.—A method of treatment which in my experience has shown many positive advantages in cases in which the requisite care can be assured, is carried out as follows: On the second or third day after operation, all gauze packing is discontinued. The nurse is instructed every two hours to syringe the ear with fresh Dakin's Fluid (*i.e.*, of not over 48 hours' standing); after syringing, the patient is to lie down and the operated ear, turned upward, is filled with Dakin's Fluid, which is allowed to remain 20 minutes. Between such treatments the ear is protected with a bit of sterile absorbent cotton in the concha (not in canal). After 3 or 4 days of this treatment, the ear should be surgically clean and free from all exuberant granulation. From this point the routine is modified as follows. Once daily the irrigation and soaking with Dakin's Fluid is carried out. Following this the bone cavity is carefully filled with chlorazine paste which is protected with sterile cotton and remains until the next daily dressing.

The above is of course nothing more than an application of the Carrell theory and treatment to the certainly not sterile bone cavity of a radical operation. This theory is based on the hypothesis that frequent flushing with Dakin's Fluid of an open infected wound will produce thorough sterilization; that chlorazine paste, though not sufficiently bactericidal to insure sterilization in the first place, may in the second be depended upon to prevent reinfection.

Chlorazine paste is made in conformity with a formula approved in the laboratory of the Rockefeller Institute, New York. It is properly prepared for the profession by the Abbott Laboratories of Chicago. Its application calls for care, and it requires a syringe, preferably glass, with a long, slender, pipette-like extremity, so curved that the paste may be delivered to any part or recess of the bone cavity.

The advantages of this treatment are: (1) Bone cavity is kept surgically clean. (2) Masses of exuberant granulations do not form. (3) Epidermization begins sooner and simultaneously in different parts of the wound. (4) The treatment is practically without pain or discomfort to the patient.

Skin-grafts.—The use of skin-grafts at the time of the primary operation is not advisable, for the following reasons,—viz.: (1) Skin does not adhere normally to bare bone; the grafts would, therefore, become effective only after islands of granulation had formed beneath them. (2) Minute foci of septic matter overlooked at the time of the operation, becoming encapsulated between the "graft" and the underlying bone, might give rise to serious local osseous necrosis. (3) Minute bone defects leading to the meninges might, under the influence of germs confined under a skin-graft, lead to intracranial infection. When, however, the cavity has become lined with a layer, however thin, of healthy granulations, the use of skin-grafts is not only safe but may hasten very materially the final healing. Usually a favorable time for applying skin-grafts is from ten days to two weeks after the primary operation.

The anterior surface of the thigh affords a convenient area from which to remove the skin. Large Thiersch grafts—as large as can be obtained—are removed with a wide-bladed razor, and slid from its surface upon specially devised spatulæ (Ballance), upon which they rest, cut surface downward, until they are introduced into the wound. In cutting the grafts, the thinnest possible layer of skin should be removed. Each graft is placed in position by sliding it from the spatula on which it rests to the surface it is intended to cover. When all the grafts are in position, small pads of sterile cotton or gauze are placed in contact with them, and pressure maintained by very careful packing of the intervening wound space.

When a single large graft has been obtained, it may be introduced in the following way: The end of a strip of sterile gauze is folded back and forth into a thick pad which is approximately large enough to fill the cavity completely; over this pad the skin-graft is folded, skin surface inward, cut surface outward. The whole is then introduced into the bone cavity and packed tightly. Unless the condition of the wound requires its removal earlier, the gauze packing should be left a full week undisturbed. When finally the gauze is removed, part or all of the grafts may remain in position, or practically all may come away, leaving, however, small islands of epithelium from which there is a rapid growth of skin over the entire cavity. The use of skin-grafts in certain cases hastens healing materially. Whether their routine use would greatly shorten the average period of postoperative repair is a question not yet settled.

In papers dealing with the after-treatment of the radical operation, much has been said of the frequent failure to obtain healing or cessation of discharge, from the Eustachian tube. To correct this condition various measures have been advised, among which is the use of small skin-grafts placed over the anterior tympanic wall, the centre of the graft being carried into the Eustachian orifice by the pressure of carefully applied gauze packing. If the tube still retains its mucosa, however, or is the seat of a suppurative process, I cannot see how this or any other non-operative measure can have any influence in bringing about permanent closure.

To epitomize: In the case of a persistent discharge from the tympanic orifice of the Eustachian tube, the writer can see only one logical method of treatment,—viz., a small, sharp curette should be introduced as far as it will go into the canal, and not only granulations, but also every vestige of mucous membrane should be removed from its lumen as far as the curette will reach. When this is successfully performed, it would seem logical to expect the formation of healthy granulations, which in so small a space should result in firm cicatricial stenosis.

No definite statement can be made as to the time in which healing will take place in any particular case. Final healing may occur in very favorable cases in from four to five weeks, or it may require ten weeks or more of constant care. Eight weeks will probably represent about the average duration of postoperative treatment before the wound is perfectly dry and lined everywhere with a covering of skin.

The Influence of the Radical Operation upon Hearing is so variable that one is never justified in making definite promises to the individual patient. Assuming the cochlea to be intact and functionating, and finding the tympanic cavity filled with granulations or the auditory canal blocked by polypi, one is naturally more hopeful as to the probable influence of the operation upon the auditory function than in a case in which such obvious obstacles to sound conduction do not exist. Not infrequently, however, very fair and useful hearing power coexists with other conditions imperatively demanding the radical operation. In such cases the hearing may be improved, may remain undamaged, and in some instances is undoubtedly reduced as a result of operative intervention. My own experience is that the hearing is oftener improved or unchanged than curtailed by the operation. The occasional cases in which serious loss of hearing has resulted from it have led to attempts to modify the operation so as to secure more uniform conservation of hearing. Of these the most widely known is that of Mr. Charles J. Heath, of London.

The Heath Operation for Chronic Middle-ear Suppuration.—This operation is based upon its author's theory that in the great majority of cases chronic middle-ear suppuration is the result of disease located within the antrum; and that the fluid products of antrum disease, draining by way of the aditus into the tympanum proper, act as constant irritants upon the tympanic and tubal mucosa, thereby perpetuating the suppurative process within the middle ear. From this hypothesis he draws the deduction that the antrum is the logical point of attack, and that, if we can eliminate the focus of disease there, the tympanic lesion will undergo spontaneous cure, or at least may be controlled by measures applied through the auditory meatus.

Operation.[6]—The first steps are precisely the same as in the radical operation. The antrum is uncovered, granulations blocking the aditus are removed by means of a curette, and the aditus is then temporarily closed by a pledget of sterile cotton. Diseased structures within the antrum and adjoining cells are dealt with in the usual way. The posterior wall and roof of the bony meatus are next removed from without inward to a point near their inner extremity, the annulus tympanicus being retained to support what remains of the membrana tympani. This brings the tympanum, or drum membrane, within easy reach, and any granulations or polypi protruding through the perforation are carefully removed. A specially constructed cannula, connected by rubber tubing with an air bulb, is then adjusted within the aditus and a forcible air-current propelled through aditus, vault, and atrium,—the purpose being to expel into the meatus any residual pus or other inflammatory products remaining within the tympanic cavity.

A plastic operation upon the membrano-cartilaginous meatus is then

[6] Heath: The Cure of Chronic Suppuration of the Middle Ear without Removal of the Drum or Ossicles or Loss of Hearing, Lancet, August 11, 1906.

performed as for a radical operation, and the postauricular wound sutured. A large rubber drain is introduced through the enlarged meatus into the antromastoid cavity. This completes the operation.

The after-treatment consists of frequent changes of the dressing and periodic inflation of the tympanum by cannula introduced into the aditus.

The advantages claimed are that the ossicles are not sacrificed, and that the tympanic membrane is frequently reproduced, with consequent preservation, and in some cases appreciable improvement, of hearing.

While the Heath operation has been tried by surgeons in all parts of the world, the results have not been sufficiently favorable to bring universal recognition of its value. Apparently it is applicable to a comparatively small class of selected cases. That it is not available in a majority of the cases for which the radical operation is employed is made clear by a consideration of the following facts,—viz.: (a) In a very large percentage of cases requiring operation the malleus and incus are necrotic, and the condition of the drum membrane beyond repair, in which case their preservation would hardly be in accord with sound surgical principles; (b) the presence of a marginal perforation, now recognized as one of the indications for surgical intervention, means partial loss of the annulus tympanicus —a condition which precludes the possibility of a regenerated drum membrane. Clearly, therefore, many of the conditions which the radical operation is designed to meet would not be relieved by the Heath operation.

Bondy's Operation.—Dr. G. Bondy, of Vienna, has also devised a substitute for the radical operation, having for its purpose the conservation of the ossicles and thereby the preservation of the hearing. Naturally it is applicable only to cases in which the ossicles are sound and the continuity of the ossicular chain intact.

Operation.—The cortex is exposed as for a radical operation, but with the difference that the membrano-cartilaginous canal is not at this stage separated from its attachment to the walls of the bony meatus. Leaving the membranous canal undisturbed insures against any injury to tympanic structures at this stage of the operation. The antrum is now uncovered and all diseased structures are removed. The aditus is enlarged so as to provide the freest possible communication between the tympanic vault or attic in front and the antromastoid cavity behind. The membranous canal is now separated from the walls of the bony meatus, and the posterosuperior canal wall chiselled away. This removal includes the inner marginal "bridge," which gives support chiefly to Shrapnell's membrane. The outer wall of the tympanic vault is carefully and thoroughly removed, this being regarded as a feature of the operation particularly essential to a successful result. The facial "spur," or ridge, formed by the lower part of the posterior canal wall, is lowered almost to the line of attachment of the membrana tensa. Granulations or diseased structures in the vault above the head of the malleus and body of the incus are removed, but the ossicles themselves are not disturbed. The operation, therefore, proposes the removal of all diseased structures from antrum, aditus, and tymapnic vault, but preserves the ossicles and whatever remains of the tense membrane. The operation is

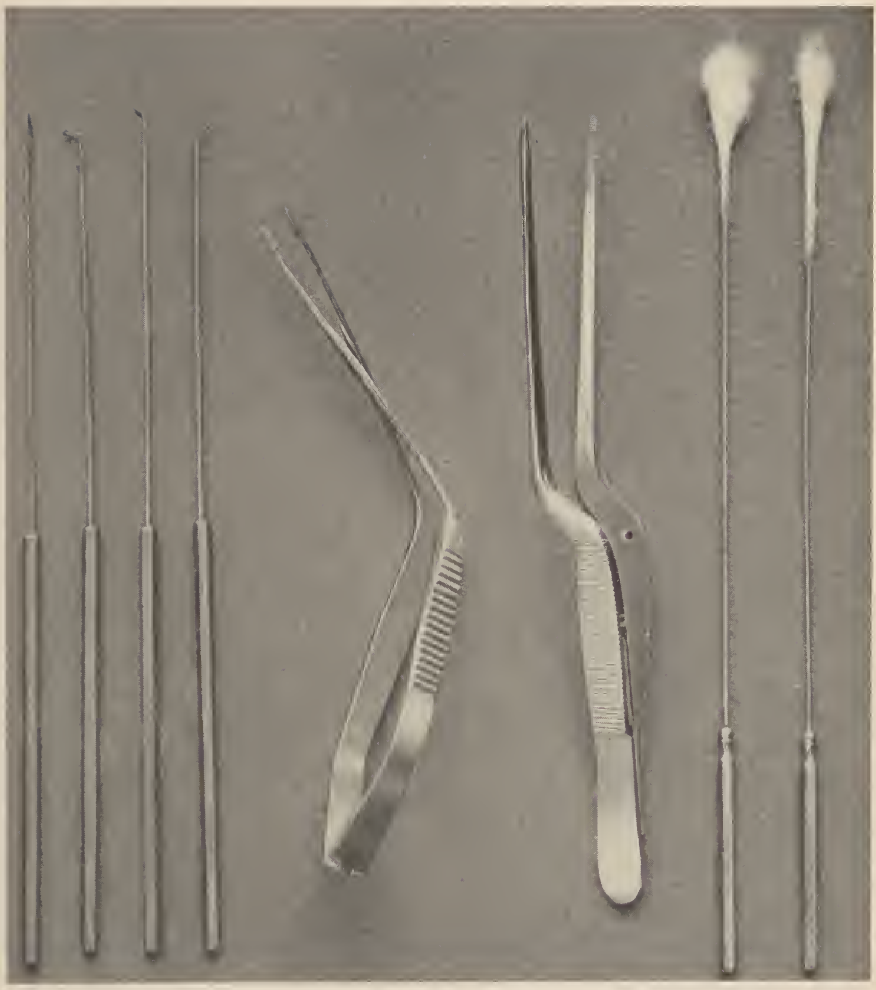


Fig. 256.—Instruments essential in the operation of ossiculectomy.

concluded, as is the usual radical, by a plastic operation upon the membrano-cartilaginous canal (Siebenmann flaps) and closure of the posterior wound.

Bondy has operated on a number of cases by this method, with successful results as measured both by cessation of discharge and the preservation of hearing. He regards it as indicated in most cases requiring operations in which the continuity of the ossicular chain can be assumed, and the hearing distance for speech in low conversational tones is two meters or over.

Streit's Operation.[7]—A similar operation has recently been proposed by Dr. Hermann Streit, of Konigsberg. Advising rather different operative technic, he secures a mechanical result which, so far as the bone operation is concerned, differs little from that of Bondy's operation,—*i.e.*, antrum and adjacent mastoid cells exposed, posterior canal wall lowered nearly to the line of attachment of the tense membrane, and outer wall of vault thoroughly removed, the ossicles and membrana tensa being undisturbed. He performs no plastic operation upon the membranous canal, which is simply replaced in position and held by tamponade. Omission of the plastic part of the operation does not appeal to the writer as promising good average results.

An interesting discussion of the relative merits of some of the proposed modifications of the radical operation will be found in a paper by Dr. Geo. L. Richards.[7a]

Ossiculectomy.—The indications for this operation cannot be reduced to exact statement. There is no possible means of determining definitely, in any case of chronic middle-ear suppuration of long standing, that areas of necrosis or foci of infection do not exist in regions beyond the reach of surgical measures confined to the tympanic vault. The value of ossiculectomy must, therefore, depend very largely upon the experience and judgment of the individual surgeon. Personally the writer has never seen a case in which he felt justified in advising it.

The most promising cases for ossiculectomy are those in which with only moderate discharge, there are evidences of ossicular necrosis,—*i.e.*, ossicles or ossicular remnants upon which bare, rough bone can be detected by the tympanic probe. Again, in certain cases in which the ossicles remain intact, their presence seems to interfere with drainage from the tympanic vault. Even in such cases, however, ossiculectomy should be undertaken only with the understanding—in which the patient should share—that a final resort to the radical operation may become necessary.

Preparation.—The auricle and parts about the ear are scrubbed with soap and water and dried. The canal should be filled with hydrogen peroxide, which five minutes later is removed by irrigating the canal and tympanum with a solution of carbolic acid, 1 in 300. The fundus of the canal—*i.e.*, tympanum—is packed with a strip of sterile gauze saturated in a 10 per cent. solution of cocaine. This is allowed to remain in position ten minutes. The tympanic mucosa, being exposed through the usually large perforation, is subject to the local influence of the drug. Removal of

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[7] Streit: Monatschrift f. Ohrenheilkunde, Bd. 45, Hft. 4.

[7a] Richards, Geo. L.: Annals Otol., Sept. 1911.

the gauze, therefore, leaves the tympanum locally anæsthetic, and the operation should be commenced at once.

THE INSTRUMENTS required (Fig. 256) are cotton applicators, ten or twelve in number, with cotton already attached; a sharp myringotome; a small angular knife; tympanic forceps; right or left incus hook according to ear operated on; tympanic ring curettes.

OPERATION.—We shall assume that any obstructing granulations have been removed at a previous sitting. Supposing the perforation to be a central one leaving a fairly wide margin on all sides, the operation is begun as follows: The myringotome is introduced anteriorly near the periphery and just in front of the short process, the cutting edge being directed downward. The membrane is then carefully incised along its peripheral margin, the knife being carried at first downward along its anterior attachment, then backward and finally upward along the posterior segment of the tympanic ring to the posterior fold (Fig. 257). The knife now cuts through the posterior fold, follows the margin of the Rivinian notch, and cuts its way forward and downward to unite with the starting-point of the original incision in front of the short process. This incision should have divided the anterior, lateral, and posterior ligaments of the malleus. The malleus may now be grasped just below the short process with a tympanic forceps, and drawn downward and removed through the canal. Should the incus have been displaced backward by this manipulation, the angular bend of the incus hook should be carried horizontally backward along the floor of the aditus, then rotated forward, bringing the ossicle with it. The incus is then grasped by the forceps and removed.

FIG. 257.—Incision lines in ossiculectomy.

The canal may be again irrigated, this time with warm boric acid or normal salt solution. It is then dried, and the canal lightly packed with a strip of sterile gauze.

Obviously the technic in removing necrotic ossicles will vary according to the condition of the ossicles, the part of the drum membrane remaining, the situation of marginal perforations, etc. When the normal connection persists, or pathological adhesions have formed, between the stapes and incus, care must be observed to avoid rupture of the stapedial capsular ligament. In such cases the small angular knife may be of use. In many cases, however, the connection between incus and stapes will have been dissolved by the pathological process which the operation is intended to relieve.

Many surgeons have recorded their varying results from this operation. One of the most interesting and instructive papers on the subject is that by Hunter Tod,[8] of London. Tod operated upon 120 cases with a result of 52 per cent. of complete cures, and 30 per cent. of the cases improved.

[8] Hunter Tod: Value of Ossiculectomy in Chronic Middle-ear Suppuration, Lancet, Sept. 3, 1910.

Plastic Operations for Closing a Postauricular Opening Following the Radical Operation. — A fortunately rare sequence of the radical operation is the presence, after healing has taken place, of a permanent opening behind the ear. This opening leads into the antrotympanic cavity resulting from the bone operation, and from its margins the skin is continuous over the entire cavity. I say the entire cavity is covered with skin, for the reason that, unless complete epidermization has taken place, the plastic operation we are about to consider is not indicated.

Mosetig-Moorhof Operation.—This operation proposes the utilization of a flap to be obtained from the tissues immediately below the opening to be closed. The entire operative field—*i.e.*, the antrotympanic cavity, auricle, and region behind the ear—is sterilized with the greatest care. Upon the skin just below the opening, a flap is marked with the point of the knife in shape conforming to, but a little larger than, the opening to be closed. Usually this flap is either oval or round in contour, and the incision takes the form of an incomplete circle or ellipse (Fig. 258). The terminal points of this incision are placed at a level 1½ to 2 mm. below the lower margin of the opening to be closed. The incision delineating this flap is carried down to the periosteum. An incision is then made around the margin of the opening except opposite the base of the flap outlined below, and from this marginal incision the skin is dissected up for about 1 mm. (Fig. 259). The flap is now dissected up from the underlying periosteum, leaving, however, its attachment at the base undisturbed. The flap is then turned upward and inverted so that its skin surface becomes part of the lining of the cavity thus closed in. The edges of the flap are sutured to the prepared margins of the opening by interrupted silk sutures or, if skin-grafts are to be used, by fine catgut sutures. The skin around the denuded area from which the flap has been taken is then elevated and drawn together and sutured (Figs. 260 and 261).

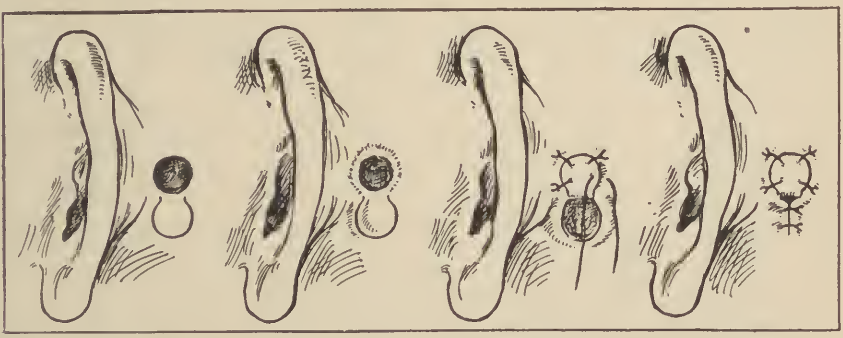

Fig. 258. Fig. 259. Fig. 260. Fig. 261.

Figs. 258, 259, 260, 261.—Mosetig-Moorhof operation, four successive steps.

Heine calls attention to the fact that the tissues immediately below the opening may be largely cicatricial and therefore give little promise of

a flap capable of re-establishing its nutrition. In such cases he has modified the operation to the extent of obtaining his flap from the tissues immediately behind the opening. This is a rational procedure, and one to be borne in mind.

Politzer states that in certain cases in which he has performed this operation he has witnessed a rather free growth of hair into the antro-tympanic cavity, this occurring in some cases in which there had been little hair in the region from which the flap was taken. The operation is, however, a rational one which should give good results in properly selected cases,—*i.e.*, those in which the opening is not very large, the bone cavity completely epidermized, and the tissues surrounding the opening healthy.

The Passau-Trautmann Operation.—The steps of this operation are clearly indicated in the accompanying illustrations (Figs. 262 to 265). Two semicircular incisions are made, one behind and the other in front of the opening (Fig. 262). The two crescentic flaps thus formed are then dissected up, the posterior from the periosteum, the anterior from the periosteum or, if the anterior margin of the opening is near the postauricular sulcus, from the perichondrium covering the posterior surface of the concha. These crescentic flaps are then inverted and their edges approximated (Fig. 263) and finally sutured (Fig. 264). These first sutures are of the finest catgut. The skin surfaces of the flaps, thus inverted, unite to form part of the lining membrane of the outer wall of the cavity thus closed in. This leaves the outer, raw surface of the flaps exposed. In order to cover in this denuded area, the surrounding skin is elevated, and then drawn together and united by means of interrupted silk sutures (Fig. 265).

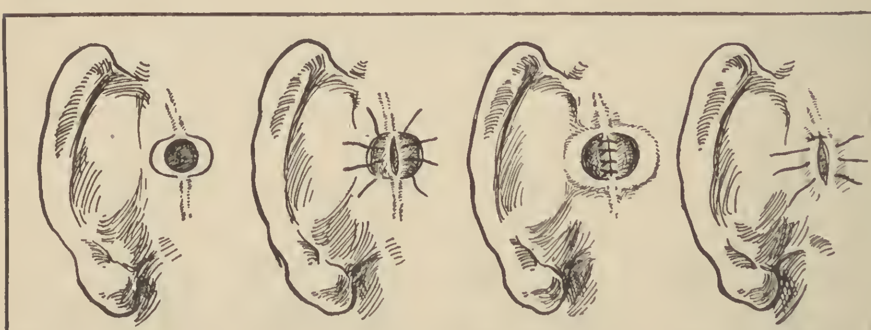

Fig. 262. Fig. 263. Fig. 264 Fig. 265.

Figs. 262, 263, 264, 265.—Successive steps of the Passau-Trautmann operation.

The operations described above should fulfil admirably their chief purpose,—*i.e.*, the closure of a postoperative opening after complete epidermization has taken place. This, however, subjects the patient to the prolonged discomfort incident to this very trying postoperative condition. In my own operative experience this particular defect has not

occurred as a result of the radical operation. I am, therefore, led to believe that except in cases of grave constitutional disease or dyscrasia it is usually preventable. By this I do not mean to imply that breaking down of the postauricular wound is always preventable, but simply that we are usually able to determine, very shortly after the original radical operation, that postauricular healing is not progressing satisfactorily, and that permanent closure is, therefore, not likely to be secured. Under such conditions I believe that this is the proper time to forestall or correct this defect, and that we subject the patient to very considerable and needless discomfort and hardship if we leave this corrective work to be taken up only after healing shall have become complete.

As aural surgeon to the Willard Parker Hospital for Infectious Diseases, I have repeatedly been obliged by the urgency of the aural symptoms to perform a radical operation upon children shortly after their discharge from the scarlatina wards of that institution. In such cases sloughing out of the sutures and imperfect union along the suture line constitute a not unusual experience. For this condition I have found reopening and resuturing of the wound quite useless, the untoward sequence of events just described being usually repeated. To meet this difficulty, I have employed a very simple operation, which I shall take time to describe briefly.

Author's Operation for Preventing or Closing Postauricular Fistula Following the Radical Operation.—Usually there is little difficulty in obtaining firm union throughout the upper and lower thirds of the postauricular wound, the point at which the wound usually breaks down being near the centre. As the operation is equally effective for a postauricular fistula of longer standing, the illustrations are drawn as for the correction of the latter condition.

The head should be shaved as for the original radical operation and the parts carefully sterilized by the usual method. The wound should be reopened by incisions conforming to the following plan: If the breaking down of the suture line is of recent occurrence, so that no considerable loss of tissue has occurred, the wound is simply reopened or incised along the recent suture line, and the edges of the lips of the wound are freshened and made straight by removing with knife or scissors the surface layer of cicatricial or devitalized tissue. If the condition is of longer standing and has resulted in a rounded opening of considerable size (Fig. 266), two incisions should be made following the lines indicated in Fig. 267. That is to say, beginning one's first incision at a point falling within the old suture line and a half inch to three-quarters of an inch below the lower border of the fistula, it is carried upward to the posterior border of the opening and thence upward to a point at an equal elevation above its upper margin. The two terminal points of this incision are next united by a second incision anterior to the first and passing through the anterior margin of the opening (Fig. 267). The skin and subcutaneous tissue included between these incisions above and below the opening are then removed. A third

incision, longer than the first two, slightly curvilinear, and roughly parallel with and three-quarters of an inch to an inch behind the first incision, is then made, passing through the periosteum to the bone. With the Langenbeck elevator (Fig. 212), the tissues are carefully separated from the bone from the long posterior incision forward to the wound which we desire permanently to close. In doing this, great care should be taken to avoid injuring the periosteum. This absolutely relieves all tension. The postauricular wound is now reclosed with interrupted silk or silkworm-gut sutures. Sterile gauze is introduced into the shallow wound resulting from the long posterior incision (Fig. 268). The dressing should be changed

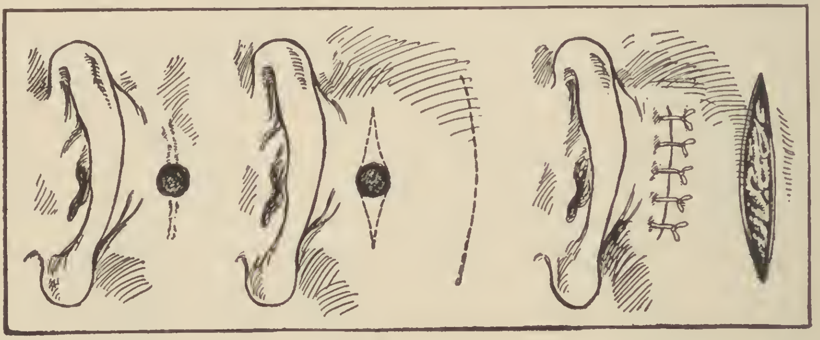

Fig. 266. Fig. 267. Fig. 268.

Figs. 266, 267, 268.—Author's operation for preventing or closing postauricular opening following radical operation.

daily. Should stitch abscesses develop, or should firm healthy union along the suture line fail, this does not foretell failure in the final result. The stitches should be removed. The posterior supplementary wound should now be used to maintain proper apposition of the lips of the wound covering the old defect or opening. This of course is done by packing a sufficient amount of sterile gauze into the posterior (supplementary) wound to push the soft parts forward and hold the anterior wound closed without sutures and without strain. This, if the dressings are carefully looked after, will result in rapid, firm closure of the wound immediately behind the ear, after which we have simply to deal with the posterior, supplementary wound, which always heals fairly quickly by granulation.

One objection which may be raised to this procedure is the scar which inevitably marks the site of the supplementary wound. If this falls within the area of hair growth, it may in a boy be made noticeable by the absence of hair in the scar line. Such a scar, if conspicuous, could be easily removed by the very simple procedure of excising the scar tissue and uniting the lips of the resulting wound.

In my hands this operation has been an effective means of preventing a permanent postauricular opening in certain cases in which it otherwise would have been inevitable.

CHAPTER XVI.

LABYRINTHECTOMY; THE RADICAL LABYRINTH OPERATION; SURGICAL DRAINAGE OF THE LABYRINTH.

Indications.—In the present state of our knowledge, there are but few conditions which may be considered positive indications for opening the labyrinth. Among these may be included the following:

(a) Chronic middle-ear suppuration with history of past attack of acute vestibular inflammation (nystagmus, vertigo, etc.); the diseased

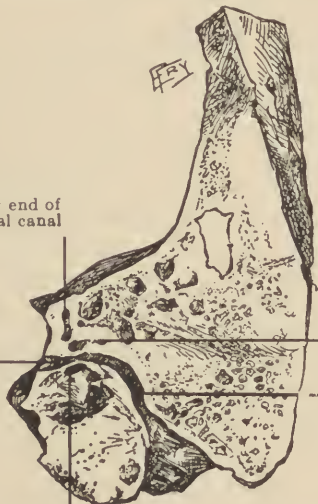

FIG. 269.—Section through tympanum, aquæductus Fallopii, and ampullar ends of horizontal and anterior vertical canals,—showing the relation of latter to oval window and horizontal portion of the facial canal.

ear being absolutely deaf and showing complete absence of caloric irritability; persistent aural discharge. This condition obviously calls for surgical relief, and, since any surgical intervention falling short of the combined radical and labyrinth operations adds to, rather than lessens, the patient's danger, the labyrinth operation must be regarded as positively indicated. It is in the latent stage of suppurative labyrinthitis that the labyrinth may be opened with least risk to the patient. Prognosis is, therefore, fairly good.

(b) Acute stage of diffuse suppurative labyrinthitis plus definite and persistent symptoms of meningeal involvement. This is a grave condition in which the patient's life is in constant jeopardy. The operation is called

for in hope of preventing further serious involvement of the meninges. Prognosis exceedingly doubtful.

(c) Symptoms of acute diffuse suppurative labyrinthitis coming on shortly after a radical or other tympanic operation, and presumably indicating an accidental rupture of the capsular ligament of the stapes. Such lesions are known to produce so high a death-rate that no time should be lost in opening and draining the vestibule. Prognosis, in early operation, —*i.e.*, before meningeal infection has occurred,—fairly good. When operation is delayed until leptomeningitis is obviously present, the chances are against recovery.

(d) Discovery during a radical operation of an open oval window (stapes being absent or the capsular ligament ruptured) through which pus exudes.

(e) Fistulæ, due to necrosis in any part of the inner wall of the atrium, —*i.e.*, vestibular windows or upon promontory,—through which pus can be seen to escape.

Among the physical conditions which in themselves constitute a reason for opening the labyrinth, a fistula in the outer wall of the horizontal semicircular canal is not included. The writer shares the opinion of those (Hinsberg, Panse, Heine, and others) who believe that a fistula in this location may, though rarely, represent a distinctly circumscribed labyrinthitis, from which the patient not infrequently makes a spontaneous recovery. In such cases the question of operation should be decided only after a careful functional examination,—*i.e.*, by the presence and character of symptoms pointing to diffuse suppurative labyrinthitis.

Before attempting to describe the various methods of operating upon the infected labyrinth, it may be well to refer very briefly to certain differences of opinion as to the mechanical results which the operation should aim to secure.

Free opening of the vestibule is the primary aim of all operations for suppurative labyrinthitis. Jansen opens the vestibule above and behind, and believes it unnecessary, unless its margins are necrotic, to enlarge the oval window downward or to remove the outer wall of the promontory. In this belief he is at variance with all other surgeons who have made suppurative labyrinthitis a special study. Hinsberg and Neumann apparently believe that by opening the vestibule freely above, and then removing the outer wall of the promontory, we secure drainage both of the vestibule and the cochlea, and that this in the great majority of cases fulfils every surgical indication. The operation proposed by Richards calls for separate exposure of each semicircular canal, free opening of the vestibule, removal of the outer wall of the promontory, and, when the cochlea is apparently extensively involved, for complete uncovering of the infected cochlear spaces. We shall return later to a discussion and analysis of these somewhat divergent views.

Surgical Guides to the Vestibule.—The following structures may be utilized as surgical guides to the position of the vestibule:

(a) *The anterior, or ampullar, end of the horizontal semicircular canal:* Removing the upper wall of this part of the canal,—*i.e.*, uncovering it superiorly,—the roof of the vestibule lies just behind, or rather internal to, its ampulla.

(b) *The posterior end of the horizontal semicircular canal:* This may with care be traced to its small opening in the posterior wall of the vestibule.

(c) The so-called "*solid angle*": By opening separately the three semicircular canals, and observing the point at which their respective planes intersect one another, we are able to deduce the position of the vestibule.

(d) *Combined use of oval window and ampullar end of horizontal canal:* The oval window having been enlarged by removal of the promontory a minute probe, the end of which is bent to a right angle, is introduced through the oval window and carried upward to the vestibular roof, which lies behind the ampullar end of the horizontal canal (Hinsberg).

(e) Approaching the vestibule from the posterior fossa (Neumann), *the posterior vertical and the horizontal canals* are opened; the line in which their planes intersect each other leads directly to the vestibule.

Operation.—Whatever operative method is adopted, the opening of the labyrinth must be preceded by a very complete radical operation. If the position of the sigmoid sinus permits, the antromastoid cavity should be enlarged backward sufficiently to allow ample room for the manipulation of instruments. The "facial spur" should be reduced to the lower limit of safety and always sufficiently to bring the stapes or oval window into clear view. Other structures which should be clearly seen are the niche of the round window, the horizontal portion of the facial canal, and the prominence of the anterior half of the horizontal canal. These represent preliminary steps in any operation for diffuse suppurative labyrinthitis. It should be said here that for the operation upon the labyrinth itself brilliant illumination, either by reflected light or, better, by the electric forehead lamp, is absolutely essential.

The Jansen Operation (intratympanic).—Jansen begins by removing the roof of the horizontal semicircular canal. In accomplishing this great care is observed not entirely to obliterate the canal or to injure its floor, since the removal of too much bone in this direction endangers the integrity of the facial canal which lies just beneath. Having uncovered the canal by removal of its superior wall, its anterior end and ampulla are used as guides to the lower limit of safety,—*i.e.*, below which the facial canal is likely to be fractured. The posterior portion of the horizontal canal, which curves backward and away from immediate proximity to the Fallopian aqueduct, he removes wholly. He now has a choice of two points of attack at which to open the vestibule,—viz.: (A) Anteriorly,—*i.e.*, to the inner, or medial, side of the ampullar end of the horizontal canal. By this route, which perforates the tympanic roof, great care is necessary to avoid fracturing the ridge of bone which includes both the floor of the horizontal canal and the horizontal portion of the facial canal. (B) The vestibule may also be reached posteriorly,—*i.e.*, by tracing the horizontal

canal in the direction away from its ampulla, and finally excavating in the direction of its small end, which opens upon the posterior wall of the vestibule. The latter course is regarded as providing greater security to the facial nerve. Having entered the vestibule by the posterior route, the opening is enlarged by the careful removal of the roof,—*i.e.*, parallel with the anterior arm of the horizontal canal and in the direction of its ampulla. This provides a free opening of the vestibule through its posterior wall and roof, and usually completes the operation.

If its margin shows no evidences of necrosis, the oval window is not enlarged downward and the outer wall of the promontory is not removed.

Hinsberg's Operation.—The preliminary radical operation having been completed, Hinsberg recommends beginning the labyrinthine operation by enlarging the oval window. The stapes if present is extracted. The enlargement of the oval window is commenced by removal of the small bridge of bone connectiong the oval and round windows. With a small curette, the window is now further enlarged by removal of the outer shell of the promontory forward and downward. A small tympanic probe with the end bent to a right angle is now introduced directly through the enlarged oval window and carried upward to the roof of the vestibule.

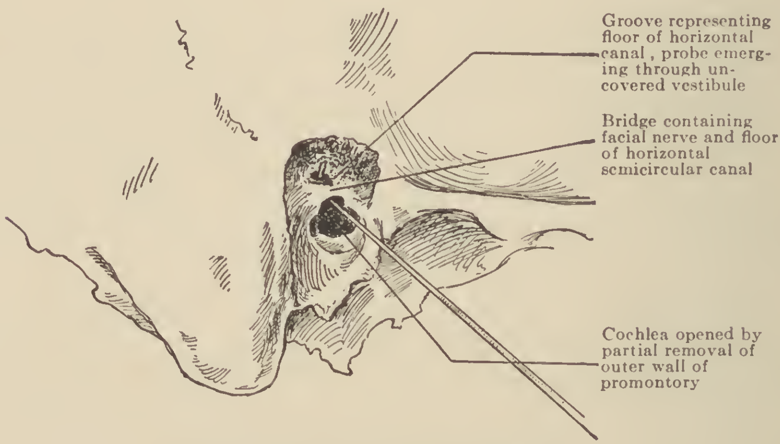

Fig. 270.—Hinsberg's labyrinthine operation.

This indicates the position and level of the roof of the vestibule. We must now remove the roof of the ampullar end of the horizontal canal, which requires the reduction of its co-called "prominence" from above downward until its lumen is exposed. This may be accomplished by shaving off thin layers of bone with a small straight-edged chisel, by means of a curette, or by the careful use of an electric burr. When the lumen of the canal is exposed, the tympanic probe may again be introduced. The roof of the vestibule, having been thus definitely located by its relation to the ampulla of the horizontal semicircular canal and by the direction of the

probe, is perforated by means of a burr or very small chisel. The opening is then enlarged backward (Fig. 270).

To summarize: The successive steps of the Hinsberg operation are (a) enlargement downward of the oval window and removal of the outer wall of the promontory; this exposes freely the lower part of the vestibule and the basal whorl of the cochlea; (b) removal of the superior wall of the ampullar end of the horizontal semicircular canal; (c) perforating the roof of the vestibule and subsequent enlargement of this opening; (d) removal by very careful curettage of diseased structures—*i.e.*, granulations, etc.—from the spaces so exposed. In the author's opinion, this is a rational, safe, and fairly complete method of draining the vestibule.

Richards's Operation.—This method proposes the separate exposure and opening of each of the three semicircular canals. When this has been accomplished, we have clearly indicated the planes of the three canals which stand at right angles to each other and enclose what is spoken of as "the solid angle." The roof of the vestibule falls within the plane of the horizontal canal. By the solid angle is presumably meant the angle formed at the point where the respective planes of the three canals intersect each other (Fig. 271). This marks the point beneath which the common tube

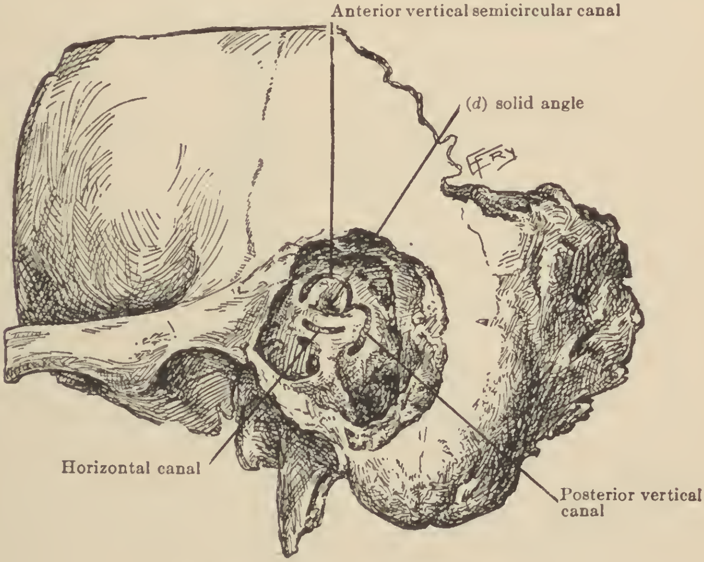

Fig. 271.—So-called "solid angle."

of the two vertical canals bends outward to enter the vestibule. It is at this point that Richards enters the vestibule, creating here a conical depression which is gradually lowered until the vestibule is reached. "The opening of the vestibule is now enlarged until a full exposure is obtained of this portion of the cavity." The remainder of the operation includes the

removal of the bridge of bone connecting the oval and round windows and the uncovering of the first cochleal whorl by removal of the outer shell of the promontory from behind forward and downward. In cases in which the cochlea has been extensively invaded, Richards advises that it be exposed and opened to the extreme limit of the morbid process.

NEUMANN'S OPERATION approaches the vestibule from the posterior fossa of the skull.

Preliminary Steps.—The radical operation is completed in the usual way, and the sigmoid sinus is in part uncovered. In front of the anterior margin of the sinus, the bone covering the cerebellar dura is removed from the tegmen antri above to the forward bend of the sinus below. The dural covering of the cerebellum is thus exposed over a triangular space which is bounded above by the roof of the mastoid, behind by the sigmoid sinus, and in front by the base, or posterior surface, of the petrous pyramid. This posterior surface of the os petrosa constitutes the operative field from which a pathway to the labyrinth is made.

The Labyrinthine Operation.—The vestibule is approached by the gradual cutting away of the posterior surface of the petrous bone. This is best accomplished by means of a sharp chisel or gouge. As successive very

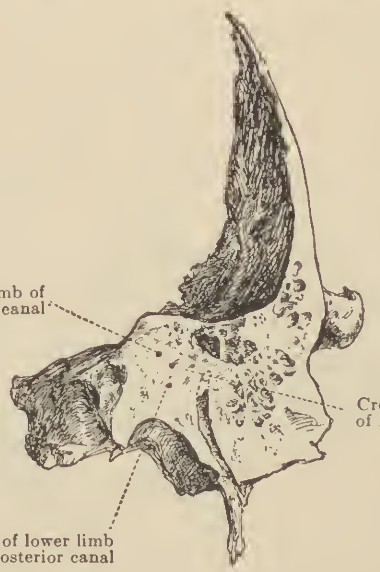

FIG. 272.—Vertical section through aditus and vertical portion of facial canal.

thin layers of bone are removed, the bone surface should be scrutinized for the first exposure of the posterior vertical semicircular canal. This may first be recognized as a short linear discoloration or, if its lumen is opened, as a short vertical tunnel representing the open canal. As the removal of bone proceeds, this part of the posterior vertical canal is also removed.

Above and below, however, are seen two small circular orifices which represent cross-sections of the canal, from which points its superior and inferior arms are continued to their termination in the vestibule. Further removal of bone brings next into view the posterior arc of the horizontal semicircular canal, its lumen lying horizontally and at right angles to the plane of the posterior vertical. When this also has been cut away, we see a third small circular orifice in the bone, lying about midway between and a little external to those first noted. This, of course, represents a cross-section of the posterior arm of the horizontal canal, which terminates in the posterior wall of the vestibule (Fig. 272).

If we imagine lines joining the three orifices so exposed, we have enclosed a slender triangular space, the centre of which will mark the point of our attack upon the vestibule (Fig. 273). Having thus determined the

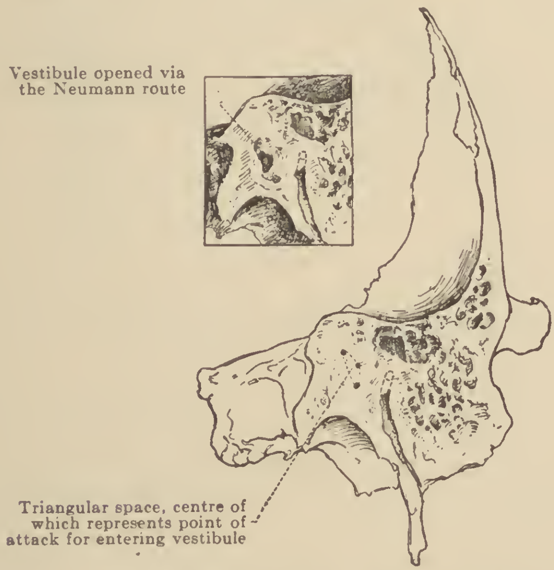

Fig. 273.—Surgical guides to vestibule (Neumann's operation).

point of attack, we have three sources of information as to the direction which the excavation shall take to perforate the posterior wall of the vestibule,—viz., (a) its known relation to tympanic landmarks,—*e.g.*, its position just behind and chiefly above the oval window; (b) the obvious direction of the open posterior crus of the horizontal canal; and (c) the estimated plane of the posterior vertical canal, the two ends of which we have open before us. This avenue of approach has the advantage of being well behind the bend and descending portion of the facial canal, so that the facial nerve is in no danger of injury at this stage of the operation.

The vestibule having been exposed through its posterior wall, the opening may be enlarged by the careful removal of the roof. The removal

of the promontory from the oval window downward and forward is carried out in the usual manner.

Comparative Advantages and Disadvantages of the Different Operations.—*Jansen's operation* is a practical method of uncovering the upper portion of the labyrinthine vestibule. As a therapeutic measure its efficacy is marred by its failure to provide drainage below by the enlargement of the oval window and the removal of the outer wall of the promontory. To this is probably due the unusually large percentage of deaths (25 per cent.) in Jansen's recorded series of labyrinthine operations.

Hinsberg's operation provides a systematic and comparatively safe method of opening the vestibule and basal turn of the cochlea which, if properly executed, can not fail adequately to drain these spaces. As compared with the Neumann operation, it offers a distinct advantage, in cases in which the disease is confined within the labyrinth, in that the sigmoid sinus and dural covering of the cerebellum are not uncovered. In execution it is simpler than Richards's operation and the surgical guides utilized in locating the vestibule are more definitely indicative of the position of that little cavity.

Richards's operation differs in its mechanical results from those of Jansen and Hinsberg in its insistence on the separate opening of each semicircular canal and the complete exploration of the cochlea when the cochlea is extensively involved. As described by its sponsor, it presents the following difficulties for the student,—viz.: (a) no directions are given for locating the two vertical canals, and their delineation is essential to the utilization of the so-called "solid angle;" and (b) the term "solid angle" is a somewhat indefinite expression which conveys no impression of the exact point for opening the vestibule.

Neumann's operation offers the following advantages over the others: (a) The risk of injury to the facial nerve is practically or almost nil; (b) in case of suspected deep-seated epidural abscess in the cerebellar fossa, this lesion is investigated and perhaps relieved by this operation; and, again, such lesions, though unsuspected, are sometimes brought to light by this method; (c) Neumann claims that the formation of healthy granulations occurs more rapidly from healthy dura than from the dense bone of the labyrinth, and that the period of postoperative healing is, therefore, actually shorter than by other methods.

The chief objection to the Neumann operation is the unwillingness of many surgeons to open the brain cavity in the absence of suspected intracranial disease, and the risk, however small, which such exposure entails. When the sigmoid sinus is placed far forward,—*i.e.*, very near the posterior wall of the bony meatus,—Neumann's operation is not practicable.

Before leaving the discussion of operative methods or technic, the writer wishes to describe very briefly a method of opening the vestibule which has seemed to possess the advantages of simplicity and comparative safety.

Author's Method of Reaching the Vestibule.—The preliminary radical operation prepares as usual the way for the surgical attack upon

the labyrinth. This leaves in view the tympanic landmarks, including the prominence of the horizontal semicircular canal (Fig. 274, *a*). Above this prominence, and between it and the tegmen tympani, is a space usually containing more or less diploic tissue (Fig. 274, *b*). The first step toward

FIG. 274.—Radical operation preliminary to opening labyrinth.

the vestibule is made by thoroughly excavating this space by means of a suitable bone curette. This leaves an open space of variable height and considerable depth above the horizontal canal and vestibule (Fig. 275, *a*). The next step is the exposure of the lumen of the horizontal canal by removal of its upper wall. If we attempt to accomplish this by means of a chisel,—even though we try to remove only the thinnest layers,—we run a very considerable risk of causing a fracture involving the facial canal, with resulting facial nerve injury. If we employ the ordinary curette, the force or pressure required in its use involves a similar danger. The writer believes that he eliminates these risks very largely by the use of a specially designed curette (Fig. 276), the edges of which are very sharp. In employing this instrument, one should depend upon the sharpness of its cutting edges and use rather rapid scraping movements, with relatively little pressure, to lower or excavate the bone from above downward. In this way the upper surface of the bony shelf containing the horizontal canal and covering the vestibule is rapidly scraped away. Sufficient force is not used to risk fracturing the bone. While this excavation is in progress, the upper bone surface is under constant scrutiny for the first signs of the horizontal semicircular canal. This is first seen as a linear discoloration, or rather loss of ivory-whiteness under the bone surface, and then, as its lumen is opened, as a very narrow

linear depression. This method of uncovering the canal has the advantage of absolute safety so far as any risk of producing a fracture into the facial canal is concerned. As soon as the canal is opened, it should be traced

Fig. 275. — Excavation preliminary to uncovering vestibule.

Fig. 276.—Author's curette.

Fig. 277.—Vestibule with roof removed.

forward by means of the same instrument to the beginning of its ampulla, and backward to the beginning of its posterior arc. This gives the physical condition shown in Fig. 277, *a*. In exposing the canal by this method, we

are simultaneously reducing the thickness of the bone constituting the roof of the vestibule, and thus rendering easier the next step of the operation,—viz., the actual exposure, or opening, of the vestibule. We locate the roof of the vestibule by its known position just behind, or internal to, the ampulla of the horizontal canal, and also its position internal to a vertical line passing through the niche of the oval window. By elevating the handle of our special curette we may now excavate the space behind the anterior end, or ampulla, of the horizontal canal and quickly enter the vestibule. In doing this we should avoid too great haste, as by exerting excessive pressure at this point we may place too great a strain upon the ridge of bone containing the horizontal and facial canals, and cause its fracture. Once the vestibule has been uncovered, it is an easy matter to enlarge the opening backward to the point, if desired, of completely removing the posterior arc of the horizontal canal. This, however, I believe, is rarely called for, and I can not see that it serves any particularly useful purpose unless we have physical evidences of an extension of necrosis in this direction.

If the stapes has been removed, we may now pass a small bent tympanic probe through the oval window and upward into our opened vestibular cavity, or *vice versa* (Fig. 277, *b*). The oval window is next enlarged and the promontory removed by methods already indicated.

The operation for diffuse suppurative labyrinthitis is admittedly a difficult one; but for the student its inherent difficulties have been both magnified and multiplied in some of the descriptions of technic which have been written.

Having outlined and compared the various surgical pathways to the labyrinth, it may be well to return briefly to a discussion of certain problems involved in labyrinthine surgery, whatever operative method be adopted.

In addition to the facial nerve, four structures or regions are said to be in some danger of injury during a labyrinthine operation,—viz., the bulb of the jugular vein, the carotid artery, the internal auditory meatus, and the modiolus.

The *jugular bulb and carotid artery* may be dismissed with a word. They are at no risk in the hands of a careful surgeon. Even when abnormally placed,—the jugular bulb projecting high into the hypotympanum or the artery being exposed through a dehiscence in the carotid canal,—they should be safeguarded by the deliberate care which should attend every step of an operation upon the labyrinth. I can think of but one source of danger to these vessels in such an operation,—viz., the use of an electric burr. One can readily imagine that, with either vessel occupying an abnormal position, this instrument might easily enter it before the surgeon could discover its malposition. This fact might find expression in a surgical rule,—viz., that *the electric burr should not be used in the removal of the promontory.*

Internal Auditory Meatus.—The dura here is firmly adherent to the

subjacent bone and is, therefore, peculiarly subject to injury. The point of a chisel entering the meatus either from the cavity of the vestibule or region of the base of the modiolus would, therefore, almost inevitably produce rupture of its dural lining. According to Richards, accidental opening of the internal meatus is likely to be followed by rapid and serious loss of cerebrospinal fluid, and places the patient in great danger of meningeal infection. The loss of cerebrospinal fluid apparently occurs only when the dural lining of the meatus is ruptured. I know of two cases—one of which came in my own surgical experience—in which the internal meatus was opened with a curette. In neither of them was there any noticeable loss of cerebral fluid. For this reason, I am inclined to believe that the chisel is an exceedingly dangerous instrument for use in the neighborhood of the internal meatus. I myself in the case referred to above opened the internal meatus with a curette, with no loss of cerebrospinal fluid and no meningeal symptoms. I agree with Richards, however, that opening of the internal meatus should be carefully avoided.

Modiolus.—There seems to be little question that the structure injury of which entails greatest risks is the modiolus. That we cannot make a thorough surgical exploration or exposure of the cochlea without removing some portion of the modiolus is made perfectly clear to us when we look at a section through the cochlea from base to apex (Fig. 278). If the modiolus has been actually destroyed by disease, it is possible that its channels have been closed by the inflammatory process in advance of the necrosis. If such destruction has not occurred we shall do well to content ourselves with providing free drainage from the vestibule and first cochlear turn, and not attempt such radical exposure of the cochlea as will throw open the innumerable little channels within the modiolus. In other words, I believe that in operating upon the labyrinth, we deal properly with the modiolus when we leave it as nearly as possible alone.

Fig. 278. — Horizontal section through cochlea.

In support of the above dogma we should bear in mind the chief purpose of the labyrinth operation, which is primarily to save life by preventing a spread of the infection to brain or meninges. Of the natural channels by which pus from the labyrinth may reach the intracranial cavity, those traversing the modiolus oftenest lead to intracranial disease. When we remove any part of the modiolus, we shorten and open wide these most dangerous pathways, and assume a risk altogether disproportionate to any advantage which may be expected from our misplaced surgical zeal.

This brings us to the question,—is drainage all that the labyrinthine operation should aim to accomplish? Personally, I believe that in the great majority of cases it is. The interior of the labyrinth differs from extra-labyrinthine parts of the temporal bone in this important particular,—viz., it contains no diploic or pneumatic structures, its walls being every-

where formed of hard, dense bone. Thorough drainage should therefore, place the labyrinthine spaces under very favorable conditions for rapid resolution. Even though the cochlea be extensively involved, it is surely safer to depend upon such drainage as is provided by removal of the promontory than to extend our exploration at the risk of opening the numerous channels within the modiolus, and thus facilitate the passage of infective matter to the meninges through what is admittedly the most dangerous pathway from the labyrinth.

I agree with Jansen that it is not necessary to open and explore separately each semicircular canal. When a fistula leads into one of the canals, the necrotic tract should, of course, be eliminated. But the walls of the bony canals are formed of very hard, dense bone, rarely becoming necrotic from within. The canals open only into the vestibule, and when the vestibule has been freely opened, adequate drainage of the canals has also been provided. That these conclusions are in accord with clinical results is supported by the fact that Hinsberg's operation, which insists only upon the opening of a single canal,—*i.e.*, the horizontal,—has in the service of the University Hospital of Breslau yielded a mortality of only 4 per cent.

In conclusion, the writer wishes to reiterate his belief that the latent stage of suppurative labyrinthitis is that during which the labyrinth can be operated upon with least risk to the patient. If, therefore, we can bring our patient through the acute stage of the disease without operative intervention, we may then operate with greatly increased chances of a successful issue. It is probably in appreciation of this ratio of the danger to the stage of the disease that Heine recommends that granulations be scraped from a labyrinthine fistula only if the symptoms of vestibular irritation have completely disappeared. In support of this contention he cites a case reported by Jansen in which "granulations were scraped out of a labyrinth which had been opened the week before, with the result that the patient developed meningitis and died." Carrying the idea still further, it would seem safer, in cases of suppurative invasion of the labyrinth following surgical injury at the oval window, to content oneself with simple drainage;—*i.e.*, having uncovered the vestibule above and the first cochleal whorl below, any curettage within these spaces should be studiously avoided as likely to determine the passage of infective matter to the meninges. This, of course, is in accord with the belief that when the latent stage is reached, the pathways of infection are to some extent closed.

Obliteration of the Vestibule for the Relief of Vertigo (Lake).[1]—Before leaving the subject of labyrinthine surgery, a word should be said of the pioneer work of Mr. Richard Lake in obliterating the membranous vestibule in cases of intractable and unbearable vestibular vertigo not dependent upon suppurative labyrinthitis. While successful operations for this purpose had previously been performed by Milligan,[2] his opera-

[1] Lake: Operation on the Vestibule for the Relief of Vertigo, Lancet, Jan. 6, 1906.
[2] Milligan: cited by Lake, ibid.

tions had consisted of the removal of the prominent external arc of the horizontal semicircular canal. We now know that, if we essay to annul the function and irritability of the vestibular nerve, it is more logical to destroy completely the structures of the membranous labyrinth, and far safer to open fully the labyrinthine vestibule than to open a single semicircular canal; for the slighter operation, to be successful, must lead to ablation of function of the entire vestibular mechanism, and it may equally as well lead to diffuse suppurative labyrinthitis should the postoperative wound by any chance become infected.

Lake's operation consists of the removal of the roof of the external semicircular canal, the tracing of this canal to its terminal openings into the vestibule, thorough removal of the roof of the vestibule, and the provision of a counter opening below the horizontal portion of the facial canal by extraction of the stapes.

This procedure, as also in some cases was the technically simpler operation of Milligan, has proved successful in attaining the chief end in view, —viz., the relief of the vertigo. The operation, while it has proved successful in Lake's hands, is indicated only in a very limited class of cases. Mr. Lake himself states that it should not be employed in the case of individuals with good hearing power, or even for those with a useful residue of hearing. In other words, he regards his operation as indicated only in cases in which the cochlear function is so far lost as to render its preservation a negligible consideration. Whatever degree of hearing the patient may possess, absolute deafness in the ear operated upon is the inevitable outcome of this operation.

CHAPTER XVII.

SURGICAL TREATMENT OF INFECTIVE SIGMOID SINUS THROMBOSIS.

Before entering upon a description of operative methods, it may be well to refer briefly to certain questions which bear directly upon the treatment of sinus disease, and which are yet open to discussion.

Until quite recently it was almost an axiom among the leading aurists of America that, when a suppurative lesion within the sigmoid sinus demands that this vessel be isolated from the general blood stream, the jugular vein should be exposed and resected from a point a little above the clavicle to a point above the entrance of the facial vein.

As opposing this view must be considered the interesting and remarkably successful series of cases reported a few years since by Dr. Crockett, and to which reference has already been made. In these cases, sixty in number, and operated upon by the various surgeons of the Boston Eye and Ear Infirmary, the vein was simply divided between two ligatures, no part of it being removed. Including within this series certain cases in which evidences of meningeal or brain lesion were present before the operation, the mortality was 16 per cent. By excluding the cases of pre-existing brain or meningeal involvement, the mortality is reduced to 9 per cent. These results are at least as good as any following jugular resection of which I have seen reports.

When in Vienna four years ago, the writer was shown several cases of sinus thrombosis in the hospital wards presided over by the surgeons of the Politzer-Urbantschitsch Clinic, which had been operated upon by simple ligation. He was told that ligation was the method there employed in cases not giving physical signs of extensive jugular thrombosis, and that the results were regarded as satisfactory. Returning to Vienna two years later, he learned that these views had not been modified.

Dr. J. J. Thompson, of New York, recently told the writer that he had in the past few years followed the Boston school in operating upon a number of cases of infective sinus thrombosis by simple ligation, the ligature being applied in many cases below the entrance of the facial, and that the results were quite as good as any he had observed from jugular resection. In a case recently operated upon by the writer, the patient's condition was so unfavorable that doubt was expressed as to whether he would survive an operation. The operation had progressed as far as the exposure of the lower end of the jugular when the patient showed signs of collapse. and the operation was ended abruptly by the resection of a small portion of the vein considerably below the entrance of the facial. In this case the resected portion was so small and so situated as to be equivalent in effect to a ligation. The patient made a fairly rapid recovery.

The above facts are at least sufficiently impressive to justify me in urging that we hold this question open to discussion and our minds receptive to further evidence.

The arguments in favor of resection as opposed to simple ligation of the jugular are based upon the fact that the intima is often found to be extensively diseased over a considerable portion of its entire length, in which case resection would seem the only adequate treatment. The removal of the vein to a point above the entrance of the facial is insisted upon as a means of preventing a reversal of the blood stream and dissemination of septic matter through the latter vessel. Upon theoretic grounds this operation seems both logical and thorough, and the results have as a whole been regarded as satisfactory.

The advantages claimed for simple ligation of the jugular vein as compared with resection are (1) its comparative simplicity; (2) the usual absence of shock; (3) absence of disfiguring postoperative deformity or scar; and, finally, its advocates claim that, through the smaller drain upon the patient's vitality and nervous force, the results are better.

Personally the writer has so far followed the practice in vogue among his hospital associates, and, with the single exception of the case above cited, has resected the vein from a point somewhat above the clavicle to a point above the entrance of the facial. While he has had under his care a large number of cases in which the outer sinus wall was extensively diseased and of others presenting symptoms strongly suggestive of sinus thrombosis, his actual operative experience has been comparatively small. He has resected the jugular vein in 14 cases, of which 4 died and 10 recovered. Three of the fatal cases were secondary to severe systemic disease, one diphtheritic and two complicating scarlet fever of severe type.

In the above statement I have tried to present impartially the two sides of this question as one still *sub judice*. As to my personal belief, a review of the subject has forced me to the conclusion that in a majority of the cases in which the jugular vein has been resected, most of my own cases being included, the simpler operation of ligation would have fulfilled the surgical indications quite as well, with less disfigurement in the resulting scar and with less danger to the patient in the considerable shock which occasionally results from jugular resection.

As to the question of whether there is any class of cases in which ligation may be distinctly preferable, it seems to the writer that a positive indication for the simpler operation may be found in cases of great systemic exhaustion in which every minute saved from the anæsthetic and operative strain adds to the patient's chances of recovery.

Before considering the operative treatment in detail, I wish also to say a word in regard to a question upon which otological literature throws little light,—viz., in what class of cases is the sinus operation alone—*i.e.*, without jugular ligation or resection—indicated? Theoretically this question is easily answered by the statement that it is indicated in cases in which the physical evidences on opening the sinus are of a circumscribed lesion situated well above the lower end of the vertical limb. But how are we to determine such limitation? Suppose, for example, that we operate upon a case in which symptoms of periodic septic absorption have been present, and that on opening the sinus we have free bleeding from the

direction of the bulb, and a demonstrable clot toward the torcular end. Such a case would seem to present ideal conditions for the sinus operation alone. But can we be sure either that a secondary infection at the bulb does not already exist, or that in the manipulation incident to the opening of the sinus, its compression below and the removal of a septic clot above, we have not sown the seed for septic thrombosis below? Or suppose that we find absolutely no physical evidences of a clot; can we from this conclude that a non-obstructive parietal clot does not exist in the bulb? Personally I feel that when we have opened the sinus to the extent necessary to explore its lumen for the presence of a suspected clot, we have incurred the risk of producing a traumatic thrombus even though none has previously existed. For this reason and those stated above, I believe that, when we have assumed the responsibility of opening a sinus on account of symptoms of septic absorption, we should in every case further safeguard the patient by ligation or resection of the jugular vein.

As to which operation should precede the other,—*i.e.*, the exploration of the sinus or the ligation or resection of the vein,—two views are held,—viz.:

I. *That the sinus should be explored first*, because (a) to tie the jugular vein in advance of the sinus operation might cause a reversal of the blood current in the sigmoid and lateral sinuses, and thus carry infective matter from a septic thrombosis in the sigmoid to the venous channels of the opposite side, or through the superior petrosal to the cavernous sinus, etc.; and (b) in case of a disintegrating clot within the bulb, the ligation of the vein before the current is checked from above may so increase the pressure upon the clot as to force infective matter into the collateral vessels outside of the skull,—*i.e.*, those communicating with the upper end of the vein,—and thus establish new foci of infection. These contentions, while theoretically correct, are not in my opinion of great practical significance.

II. *That the jugular vein should be ligated or resected before the sinus is opened*, for the reason that, if the reverse order is observed, the manipulation of the sinus, and particularly the use of compresses, may dislodge and force into the general circulation portions of an infected clot which otherwise might be removed through the sinus wound.

Ordinarily, I do not believe that it greatly matters which operation is performed first. When, however, the patient's condition is so profoundly septic and his vitality so greatly reduced that the addition of any fresh septic material to that already circulating in the blood might turn the scale unfavorably, I should say that ligation of the vein should precede the opening of the sinus.

In any case, and whichever order of precedence is observed, the sinus should always be freely exposed and ready for exploration before the vein is operated upon.

Sinus Operation for the Removal of an Infected Clot.—In describing this operation, we shall assume that a very complete preliminary mastoidectomy has been performed. In mastoid surgery it is a technical rule that the margins of the bony wound should, as far as may be, present shelving surfaces,—*i.e.*, its posterior boundary should not present a sharp

edge of cortex leading precipitately into a deep cup-shaped cavity. When the sinus is to be exposed, it is particularly important that the cortex should be freely removed behind the sinus groove, since the attempt to uncover the sinus at the depth of a narrow bony depression would be both difficult and dangerous. Obviously, it would be neither easy nor safe to attempt the exposure of the sinus until its bony groove had been clearly outlined throughout the greater extent of the mastoid cavity. This usually, though not invariably, delineates the vessel's course from the knee, or bend, above to the point at which it curves forward below to enter the jugular foramen. The preliminary mastoid operation, properly executed, should leave a bony cavity, or excavation, somewhat resembling that shown in Fig. 279.

For the initial removal of bone from the intact sinus groove, a flat chisel of moderate width is undoubtedly the safest instrument. Usually it is more convenient to remove the first layer of bone from behind forward rather than vertically,—*i.e.*, along the course of the sinus. The chisel

Fig. 279.—Bone operation preliminary to exposure of sigmoid sinus.

Fig. 280.—Position of chisel in uncovering the sigmoid sinus.

should be held as slantingly as possible,— *i.e.*, with its flat surface lying as nearly flat upon the surface of the sinus groove as will allow its edge to engage the bone (Fig. 280). Only the slightest taps of the mallet should be used. Employed in this way the chisel penetrates the bony plate of the sinus groove, but practically never injures the dural coat of the vessel, unless the latter is actually adherent to the overlying bone. The latter condition is usually indicated, however, by the macroscopic appearance of the bone, and will be further indicated by its softness and the ease with which the chisel enters the bone. When the edge of the chisel has passed beneath the sinus plate for a little distance, it is withdrawn slightly and then, by cautiously lowering the handle, the first scale of bone is gently prized upward and removed, exposing the dura beneath. By repetition of this process—using always the greatest care—the first exposure of the sinus is gradually enlarged. When we have uncovered the sinus over an area approximately half an inch in length and of sufficient width to admit of the introduction of instruments beneath the bone, it is usually safer and easier to lay the chisel aside and substitute other instruments. If now we

can introduce a curved round-edged separator between the sinus and the overlying bony plate, showing absence of adhesions, we are in a position to use some form of rongeur with advantage.

Fig. 281.—Rongeur.

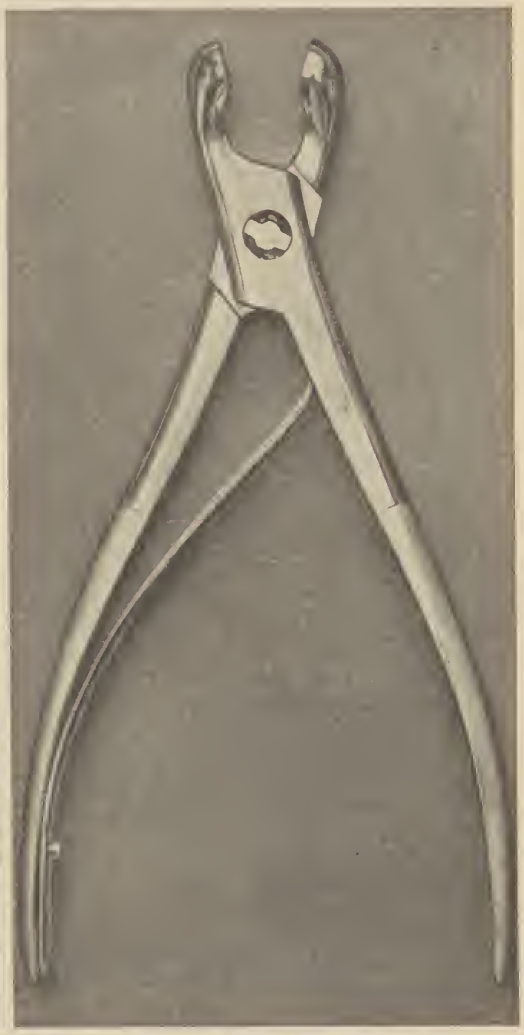

Fig. 282.—Rongeur.

Use of the Rongeur.—For uncovering the sinus the rongeur is either a safe or a very dangerous instrument according to the type of instrument and the way it is used. A sharp, thin-bladed rongeur (Fig. 281) is a dangerous instrument for this particular work, on account of the ease with which its edge may engage a hidden dural surface. Fig. 282 shows a

rongeur the thick, rounded cutting extremity of which may be actually employed to separate the sinus wall from its bony covering. As to the method of using the rongeur for exposing the sinus, if one blade is made to engage the edge, or margin, of the bony opening, and the other is made to engage the cortex further back, this cortical point being used as a fulcrum, the exposure may be quickly enlarged, but there is always danger that the lower blade may slip slightly and the sinus wall be caught between its edge and the bone to be removed. A much safer method is by using the comparatively thick-bladed rongeur (Fig. 282), the lower blade of which is introduced between the sinus wall and overlying bone, and made to do actual duty in separating the one from the other. By slightly withdrawing the instrument just before the blades are brought together, there is little danger of opening the sinus.

The objection which has been advanced to the use of the rongeur,—viz., that the introduction of any instrument between the bone and sinus wall may by its displacement of the latter dislodge a parietal clot,—is, of course, theoretically tenable. It is, however, a purely theoretic contention, since such an accident is not known ever to have occurred, nor does it seem mechanically probable.

The sinus should be exposed from the knee to the lowest point before it bends forward to disappear within the jugular bulb (Fig. 283).

The various macroscopic changes occasionally observed in the outer sinus wall have been described in a foregoing chapter, and need not be dealt with here. The sinus has been found to contain a septic clot in cases in which the outer dural coat was of normal appearance; and, *per contra*, it is not uncommon to see the outer coat covered with granulations or showing other pathologic changes while the interior of the vessel remains quite free of disease. We are not, therefore, able to determine from the physical signs alone the presence or absence of an infective clot within the sinus.

FIG. 283.—Exposure of sinus wall preliminary to its exploration.

In preparation for the opening of the sinus, the entire wound should be flushed out with sterile normal salt solution. Fresh sterile towels are placed about the head and around the wound. The surgeon's hands are re-sterilized, or, if operating in gloves, these are changed. All instruments used in the preliminary operation are discarded or re-sterilized,—*i.e.*, only freshly sterilized instruments should be used for the intra-sinous operation.

The instruments especially needed for exploring the sinus are an ordinary scalpel (kept in alcohol until needed), flat scissors and scissors curved

on the flat, both sharp, two ordinary thumb forceps for holding iodoform plugs, bayonet forceps, toothed forceps, and one or two dull ring curettes. There must also be in readiness a number of gauze plugs—for safety, say a dozen—consisting of small, tightly rolled bundles of iodoform gauze, about three-eighths inch in length and of the diameter of a small lead-pencil. These are very essential for the control of hemorrhage (see Fig. 235).

In addition to the above, there must also be at hand mallet and chisels or rongeurs, scalpels, periosteal elevators, artery-clamps, etc., for use in case the conditions within the sinus should call for further removal of bone, —*e.g.*, for the exposure of the lateral sinus from the sigmoid bend backward toward the torcular.

Exploration of the Sinus.—In this operation our aim is to expose the interior of the vessel to actual inspection. To attempt less but adds to the patient's risks, and supplies the surgeon with absolutely no data of diagnostic value. It is not sufficient, therefore, to puncture the vessel, or even to make a short incision in its outer wall over that region in which we believe infection most likely to have occurred. To arrive at any practical results, either diagnostic or curative, we must incise the outer wall throughout approximately the entire extent of the portion exposed. We must be prepared, therefore, to control hemorrhage promptly and effectively as soon as the sinus is opened.

An assistant picks up upon the points of ordinary thumb forceps two of the small iodoform plugs, one in each hand. These are held at the upper and lower ends of the exposed sinus, but pressure is not exerted until the sinus wall has been incised (Fig. 284). With a scalpel the surgeon now makes a careful incision—about half an inch long—in its outer wall about midway between the compresses. This incision is later extended in both directions by means of sharp blunt-pointed scissors. Unless its lumen is actually filled by an occluding thrombus at the site of the incision, or by thrombi above and below, the incision is followed by a gush of blood. This is allowed to continue for a few seconds in order to gauge roughly its volume. This free outpouring of blood does not signify that a clot does not exist either toward the torcular or toward the jugular end of the vessel, for in either position a parietal, non-occlusive clot may be present; and again, even with an occluding thrombus toward the torcular end of the vessel, the return flow from the inferior petrosal sinus through the jugular bulb will cause very copious hemorrhage.

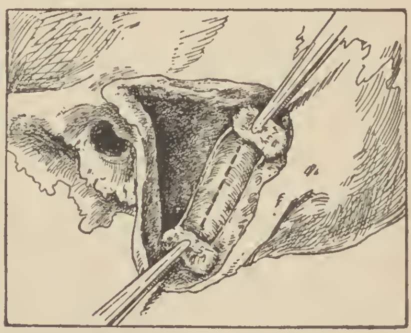

Fig. 284.—Position of gauze plugs for controlling hemorrhage when sinus is opened.

A few seconds after the sinus is opened, the assistant exerts pressure

upon its upper (torcular) end. If by this procedure the flow of blood is absolutely controlled, we know that the lower end of the sigmoid sinus or the jugular bulb is completely occupied by an occluding thrombus; if only a little blood trickles through, we know that the jugular end of the sinus contains a clot which only partially occludes its lumen. If compression of the vessel above exerts no influence upon the hemorrhage, we know that the jugular bulb is not occluded, but we do not know—nor is it possible to determine—that an infective parietal lesion does not exist in that situation. The assistant now exerts pressure upon the lower, or jugular, end of the sinus. This, as a rule, effectually controls the bleeding; or, if bleeding continues after compresses have been effectively applied above and below, we know that the blood must come from the mastoid emissary entering the sinus between them. Let us suppose, however, that all bleeding has ceased. The assistant now removes the compress from the upper end of the sinus, this, if no clot is present in the lateral sinus,—*i.e.*, between our incision and the torcular,—being followed by a renewal of the hemorrhage. Should the removal of pressure from the upper end of the exposed sinus be followed by no bleeding, this fact would announce to us that the hemorrhage had from the first come solely from the direction of the bulb, and from this we would deduce the existence of an occluding thrombus in the lateral sinus somewhere between the sigmoid bend, or knee, and the torcular.

It is possible, then, from the simple surgical measures above described—which, however, require some care and surgical skill in their execution—to determine the presence of an occluding thrombus (a) at the site of the incision, (b) at the jugular end of the sigmoid or within the jugular bulb, or (c) at some point within the lateral sinus between the upper exposure of the sigmoid and the torcular. Or, by an obviously diminished flow of blood from either end, it is possible (but exceedingly rare) that we may be able to locate a parietal and only partially occluding clot in one or the other situation. We must now outline very briefly the further surgical treatment indicated by these various conditions.

I. Supposing, for example, that cessation of hemorrhage following compression at the lower, or jugular, end of the sigmoid, demonstrates the presence of an occluding clot somewhere within the lateral sinus: we can not from this determine whether the clot is just behind the knee or situated at or near the torcular. If we attempt to determine this point, and at the same time dislodge the clot, by inserting a curette into the sinus and backward in the direction of the torcular, we may fail of our purpose and yet inflict injury upon the inner wall of the vessel, leading later to a suppurative lesion within the brain itself. It is a more surgical, and therefore a safer, procedure to uncover carefully the sinus from the bend backward toward the torcular, splitting the outer wall of the vessel with a scissors as we go. Naturally there will be no bleeding until the thrombus is reached. It can then be removed cleanly and without injury to deeper parts, and the vessel plugged. We then proceed to cut away with scissors the outer wall of the sinus on either side of the incision throughout its

entire extent. This removes the possibility of pus or other infective matter collecting within the lumen of the open and now useless sinus, and creates the most favorable condition for a healthy granulating wound.

Since the presence of a septic clot near the torcular is no proof that the jugular bulb is not the site of a second infective thrombus, and since also the very opening of the vessel may have paved the way for infection below, I am personally in favor of further safeguarding the patient by ligating or resecting the jugular vein.

II. Should absence of hemorrhage from the jugular end of the sinus indicate the presence of a thrombus completely filling the jugular bulb, or should a greatly diminished blood flow point to a partly obstructive parietal clot, I am inclined to believe that we should make no attempt to dislodge it until the jugular vein has been ligated. The inferior petrosal sinus is presumably still patent, and the attempt to introduce a curette into the region of the bulb, if successful in dislodging a clot there, is altogether more likely to force it into the general blood stream than to effect its removal through the opening in the sigmoid sinus. After the jugular is tied, we may make a careful attempt to dislodge the clot from the jugular end of the sigmoid, since the blood stream from the inferior petrosal and the posterior condyloid vein would then be in reversed direction,—*i.e.*, toward and through the opening in the sinus wall.

III. Supposing that our incision of the sigmoid sinus demonstrates the presence of a parietal clot limited in its extent to the site of the incision; or, again, supposing that free bleeding occurs from either end, and that there are absolutely no physical evidences of a clot or infective lesion in any part of the sinus, these negative findings constitute no proof that a focus of infection is not present within the bulb or even at the upper end of the jugular vein. The logical treatment is, therefore, to compress the sinus above and below, cut away its outer wall throughout the entire extent or greater part of the portion exposed, pack the wound with iodoform gauze, and then ligate or resect the jugular vein.

I have more than once resected the jugular vein in cases in which no positive physical signs of a clot within the sinus could be found, with prompt relief of all symptoms of periodic septic absorption.

Resection of the Jugular Vein.—The rationale of this operation is based upon the theory—so far correct—that the elements of infection may involve not only the sigmoid sinus and jugular bulb, but also the walls of the jugular vein far below its point of exit from the jugular foramen. Hence, it is assumed that safety demands in every case its complete removal. The correctness of this latter hypothesis is by no means so clearly established.

Preparation.—The side of the neck, from the region of the mastoid tip above to the clavicle below, is shaved and thoroughly cleansed. In scrubbing this area, all deep pressure or unnecessary manipulation along the course of the vein should be avoided, as by this means we might easily dislodge a septic clot, which would of course be thrown into the general

blood current. The cleansing should, therefore, be accomplished with the least possible disturbance of the deeper structures. Possibly shaving and subsequent painting of the operative field with tincture of iodine may prove the ideal preparatory treatment for this particular operation.

The patient is placed upon his back, with the head turned so that the face looks directly away from the shoulder corresponding to the diseased ear. A sand-bag wrapped in sterile towels or, if we must improvise, a sheet many times folded and rolled into a stout pad, is placed beneath the shoulders and neck. This places the sternomastoid somewhat on the stretch and also brings the deep structures concerned in the operation into more accessible and convenient position.

Operation (Figs. 285, 286).—The surgical guide for the incision is the anterior border of the sternomastoid muscle. The incision begins, therefore, at a point corresponding to the mastoid tip, or just in front of it, and follows the anterior border of the muscle downward and forward to its clavicular attachment. This incision divides the skin and platysma myoides muscle, and exposes the anterior border of the sternomastoid. In some cases the external jugular is large and its position such as to require division between two ligatures, in other cases it is possible to avoid it, and in others it is so small that the bleeding is easily controlled by artery clamps and may not require ligatures. The sternomastoid having been thus exposed to view, it is not a difficult matter to introduce a blunt instrument beneath its anterior border and deflect it from the fascia upon which it lies. This may be easily accomplished with the handle of the scalpel or even with the finger. When the muscle has been deflected throughout the extent of the incision and drawn backward, the deep fascia is brought into view. From this point, it is necessary to work with care and mostly with blunt instruments. Having divided the deep fascial layer exposed beneath the sternomastoid muscle, we come quickly upon the "common sheath" enclosing the jugular vein, carotid artery, and pneumogastric nerve. Even before this sheath is opened, the vein is usually easily identified by its large size, its prominent position,—*i.e.*, external to the artery,—and by its ballooning out with blood when pressure is applied below. Before attempting to open the sheath, it should be exposed well throughout the length of the incision by deepening the wound in its upper portion, where the vessels lie more deeply. The sheath is then opened at the lowest point exposed in the wound. This is most

Fig. 285.—Line of incision for jugular resection.

easily accomplished by grasping it at two closely approximated points by means of two ordinary thumb forceps—great care being taken that only the sheath is caught—and tearing it laterally. Once opened, it is not

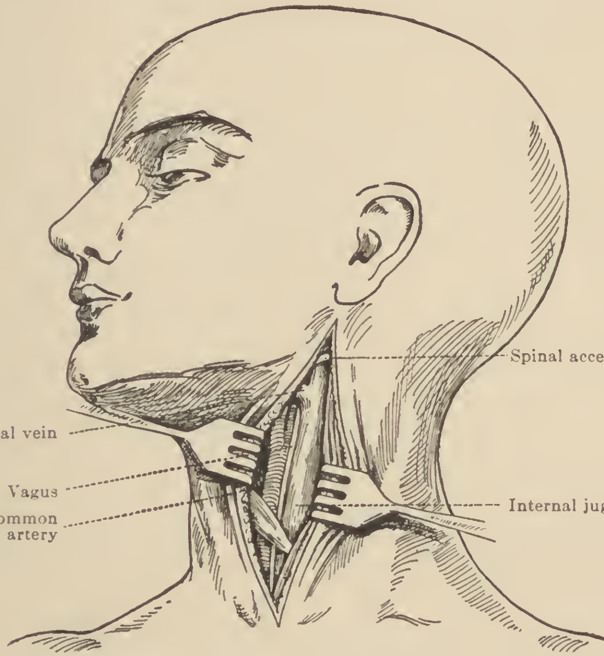

Fig. 286.—Internal jugular vein (showing relative position of vein, artery, and nerve).

difficult to introduce a blunt instrument—preferably a closed blunt-pointed scissors curved on the flat—and extend the opening as far upward in the neck as the sheath is exposed.

The next step is the separation of the vein from the artery and nerve. This may be effected by means of any blunt instrument. In the lower part of the incision, the jugular vein is external to and behind the common carotid artery, the vagus lying more deeply between them. It is obviously of paramount importance that these structures should be definitely recognized in order that the ligature which is to surround the vein shall not include the vagus. Having separated the vein from its companion structures, two catgut ligatures are passed around its lower part and tied, and the vein divided between them. The ligature is cut close upon the lower segment of the vein, which is allowed to fall back into the wound. The ligature holding the upper segment is, however, often retained for use as a retractor. This is given to an assistant, who, by gentle traction in slightly different directions, may aid considerably in separating the vein from surrounding structures. As the vein is isolated from below upward, any

branches met with are tied between two ligatures and divided. Very frequently the lower branches—*i.e.*, the middle and superior thyroids—are so small as to be overlooked and torn, in which case the hemorrhage is usually insignificant and easily controlled. The common branch of the lingual and facial veins is, however, a vessel of considerable size, which must be divided between two ligatures. The occurrence of the facial and lingual veins as separate branches springing directly from the jugular is exceptional.

It is important that the upper ligature upon the internal jugular should be at a point above the facial branch; above the facial branch I do not believe, however, that any material advantage is gained by trying to follow the vein as nearly as possible to its point of exit from the skull.

If the jugular is tied below the point of entrance of the facial, the result in many cases will be the control of the disease. There is always present, however, the possibility that septic matter—possibly parts of a disintegrating thrombus within the bulb—may be washed downward by the blood stream from the inferior petrosal, which, finding its way blocked by the ligature lower down, may be diverted through the common faciolingual branch into other channels, the bacteriæmia being thus perpetuated.

When all the branches encountered have been ligated, the jugular itself may be tied at a point above the facial, and the resected portion removed. After its removal, the wound in the neck may be flushed with normal salt solution, and its edges approximated and partly sutured, the wound being left open above and below, however, for the insertion of gauze wicks.

The mastoid wound is now again uncovered. The gauze plug is removed from the lower end of the sinus, and, if a clot is suspected of being present at the point of its entrance into the bulb, a careful and tentative attempt may be made to dislodge and remove it. If free bleeding is continued from the inferior petrosal, a gauze plug must again be applied. If absence of hemorrhage points to the presence of clot within the bulb, an attempt may be made to dislodge it from its deep position. It must be remembered however, that, even if we should succeed in dislodging it, the reformation of a clot is under the circumstances inevitable. A wick of folded gauze is introduced into the bulb, and the rest of the wound packed as after a mastoid operation.

Usually the gauze compresses used to control hemorrhage may be removed on the fourth or fifth day after the operation. If no bleeding follows their removal, it is not likely to recur later.

Obviously complete removal of the jugular is the only operation open to us when this vessel is occupied through a considerable portion of its length by an organized and presumably infected clot.

Ligation of the Jugular Vein.—But little need be said as to the technic of this comparatively simple operation. As the purpose is to ligate the vein above the facial, it is obviously unnecessary to make the long incision employed when the vein is to be exsected. An incision beginning

at, or in front of, the mastoid tip and extending downward along the anterior border of the sternomastoid muscle for a distance of two inches should be amply long. If for any reason, as in a girl or young woman, it is desired to make the incision as short as possible, it will be found better not to attempt to deflect the sternomastoid, but rather to cut directly through the muscle, beginning at a point corresponding to the centre of the mastoid tip.

When the jugular has been exposed and isolated at the point at which the facial vein is given off, one or both vessels may be tied. If the purpose is merely to cut off possible infection from a sinus lesion considerably above the jugular bulb, there being no question of disease directly involving the jugular itself, ligation of the jugular above the facial vein, and without disturbing the latter, would seem to furnish full protection from further infection. When, however, the sigmoid lesion is near to or within the jugular bulb, and there is some doubt as to whether the infection may not have involved the walls of the jugular vein below the facial, the ligation of both vessels would seem to be indicated. These, however, are somewhat theoretic considerations. When it is necessary to ligate the jugular, the additional tying of the facial vein seems so devoid of untoward results, that most surgeons will be better satisfied—probably with good reason—to have tied both vessels.

Dr. George L. Tobey, of Boston, has operated successfully upon a number of cases of infective sinus thrombosis in which he exposed the vein by a very short incision having no apparent relation to the course of the sternomastoid fibres. Tobey's incision through the skin is made parallel with some natural fold or crease of the neck, and follows therefore a direction which crosses the course of the vein. Speaking before the Otological Section of the New York Academy of Medicine, he showed a number of photographs of patients operated upon by this method, in whom the resulting scar seemed hardly noticeable. Certainly from the stand-point of cosmetic effect, his results represented a notable improvement upon those obtained by the older method.

When simple ligation of the jugular is spoken of, I assume that this means the division of the vein between two ligatures. Having decided upon interruption of the blood flow at a certain point, there is obvious reason for providing that, should the ligature break down and a suppurative lesion be there established, there shall be no chance of this fresh focus of infection influencing the general blood stream.

CHAPTER XVIII.

SURGICAL TREATMENT OF INTRACRANIAL LESIONS (CONTINUED): TEMPOROSPHENOIDAL AND CEREBELLAR ABSCESS; OTITIC MENINGITIS.

Surgical Treatment of Brain Abscess.—Up to twenty years ago most cases of brain abscess ended fatally. Bezold,[1] writing in 1906, was able to say that all the brain abscesses which he had opened had recovered,

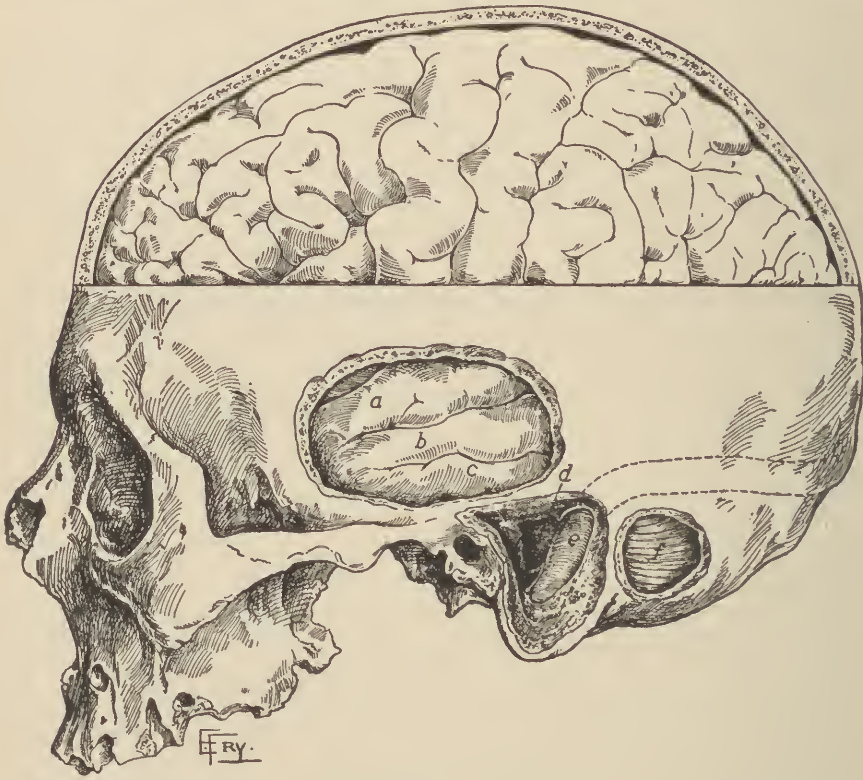

FIG. 287.—Relations of brain to temporal bone: *a*, *b*, and *c*, first, second, and third temporal convolutions.

—a fortunate surgical experience to which few surgeons can lay claim. The tendency to record successes and forget or ignore failures is so human a trait that it is difficult to arrive at correct percentages of deaths and recoveries following operation. There is no question, however, that the

[1] Bezold: Text-book of Otol., English Edition, p. 240.

average results of aural brain surgery have been vastly improved during the last decade, and I believe that the mortality from otitic brain abscess is destined to a further material reduction.

That there are differences of opinion as to the proper surgical treatment of brain abscess is shown by the diversity of methods in use. This diversity is not altogether a result of conflicting views as to how best to arrive at a given mechanical result; to some extent it is the expression of widely different views as to the tolerance of the brain to surgical manipulation.

The writer personally believes that, of cases of brain abscess which end fatally in spite of surgical intervention, more deaths are due to a faulty conception of what the surgeon is called upon to attempt than to a lack of operative skill. Before considering the vexed question of operative technic, it may be well, therefore, to refer briefly to certain facts bearing upon the scope and limitations, as well as the dangers, of surgical intervention.

In whatever part of the brain the abscess may be located,—*i.e.*, whether in the temporosphenoidal lobe or in the cerebellum,—the main purpose of operative intervention is to evacuate pus and provide for subsequent drainage. Even when this has been accomplished, however, there are certain dangers inherent in the operation itself. Chief among these are (a) direct infection of the meninges, (b) the development of a cerebral hernia, and (c) intracerebral injuries leading to fresh foci of infection.

Cerebral Herniæ.—Small herniæ occasionally contract and disappear under proper treatment. Experience has shown, however, that the development of a cerebral hernia of any considerable size in cases of otitic brain abscess usually foretells a fatal result. The patient may be up and about, and for weeks may appear to be progressing satisfactorily toward recovery. The protruding cerebral mass prevents local repair, however, and subjects the meninges and subdural spaces to constant risk of infection. The change for the worse is usually sudden, and is not the direct result of the hernia itself, but of the diffuse purulent leptomeningitis to which it in most cases sooner or later gives rise. This fact is worthy of most serious consideration, since cerebral hernia is to a very considerable extent a preventable condition which may be guarded against by proper treatment of the dura at the time of the original operation.

Dural Incision.—In the first place, it should be recognized that there is a class of cases in which no incision of the meninges may be necessary. Dean[2] proved by the results in his series of cases that a brain abscess drained through a "stalk," or pre-existing sinus, does not usually require further opening or even the introduction of drainage tubes or wicks. This fact is embodied in a rule or dogma by Heine,[3] to the effect that "neither puncture nor exploratory incision is necessary when the pus has already perforated the dura and can be seen flowing out through a fistulous opening." The truth of this statement seems a logical deduction from the con-

[2] Dean: Operative Procedure for Brain Abscess of Otitic Origin, Annals of Otology. vol. xix, No. 3, pp. 541–556.

[3] Heine: Operation on the Ear, English edition, p. 182.

ditions present; for, with such a fistulous opening, we may assume that the abscess has existed long enough for the formation of a limiting membrane, and also that the contained pus is not under pressure. Rupture into a ventricle is, therefore, not likely to occur. And, again, the adhesive processes about the dural outlet will usually have closed the pathways of infection to the subarachnoid space. The development of leptomeningitis is, therefore, equally improbable. If we attempt to enlarge the opening, we create gateways of infection. If we are content to facilitate drainage by free removal of bone about the dural fistula, we place the patient in the best possible condition for the gradual shrinkage of the abscess cavity with ultimate healing.

Körner,[4] in an analysis of the gross pathologic changes in a series of 100 brain abscesses, found in 42 cases a fistulous tract directly connecting the abscess cavity with the original suppurative focus within the temporal bone. In 15 others the brain substance was broken down between the abscess cavity and the dura, so that only the dura separated the fistulous tract from the diseased bone.

When no fistulous tract exists, incision of the dura becomes necessary. If pathological changes in the dura at some particular point indicate the position of a superficial or subdural abscess, the dura should be incised at this point. When no such dural changes are present, I believe that the dura covering the lateral, or convex, surface of the temporal lobe rather than its under surface—*i.e.*, the dura over the tegmen antri et tympani—should be incised, for the reason that the knife carried horizontally into the brain explores a larger portion of the suspected region than when it is passed vertically upward from the region of the removed tegmen. A short vertical incision through the dura—little longer than the width of a narrow knife-blade—is long enough for exploratory purposes, and incurs no risk of cerebral hernia. The practice, once rather general, of lifting semilunar flaps of membrane in order to expose the brain cortex beneath is happily falling into disuse. Aural surgeons now recognize the fact that with brain lesions of otitic origin this is not necessary, that it paves the way for unmanageable cerebral herniæ, increases enormously the risks of meningeal infection, and does not materially aid the surgeon in locating the seat of the disease. I have seen some cases of brain abscess drained after the elevation of a dural flap. All ended fatally.

As to the exploration of a cerebral abscess cavity, it is time that we awakened to the fact that any unnecessary disturbance or displacement of brain tissue is a technical fault likely to be paid for in terms of human life. The practice, once more or less common, of introducing a finger into the abscess cavity for the purpose of determining the character and extent of its limiting walls, is as illogical as it is dangerous to the patient. If the cavity is lined by a distinctly limiting membrane, no therapeutic advantage is gained through the information obtained by this method of examination; and if no limiting membrane be present, the surgeon will not be able to

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[4] Körner: Die otitischen Erkrankungen des Hirns, etc., 1896, p. 97.

distinguish the infected from the surrounding area of healthy brain tissue. Furthermore, the introduction of the finger may easily under the latter conditions determine a spread of the infection to parts not hitherto involved. In the writer's opinion, digital examination can not, therefore, be too strongly advised against.

Ballance states that, in cases of brain abscess in which the exploratory incisions have failed to release pus, the finger carefully introduced will frequently detect an encapsulated abscess as a tense, unyielding mass, which may then be opened, the finger being retained in position to guide the knife. I can conceive that this use of digital examination might in some cases serve a useful purpose, though it would seem to me a safer method to continue the search by multiple incisions or punctures.

The point I wished to make, however, is that after a brain abscess has been demonstrated and partially evacuated by a flow of pus along the knife-blade, the introduction of the finger into the abscess cavity is a useless procedure which may inflict irreparable injury upon the brain tissues.

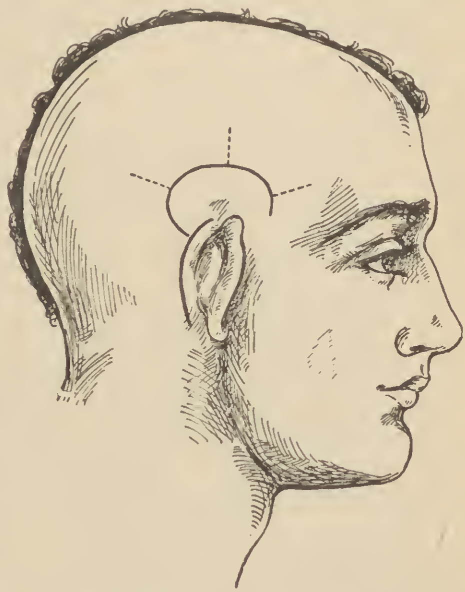

Fig. 288.—Incision for exposing squama, preliminary to an exploratory operation for brain abscess. Upper semicircular incision usually provides adequate exposure of squama. The three dotted lines, usually uncalled for, give wider exposure if required.

Operation.—In preparing the patient for an exploratory operation upon the brain, one-half of the head should be shaved (Fig. 288), and the shaved area should be cleansed and sterilized by the methods already described.

Frequently the mastoid will have been opened previously, so that we have only to scrape out and cleanse the old wound and proceed to the surgical treatment of the brain lesion. If no previous operation has been performed, the mastoid should be operated upon,—a radical operation being performed if the suspected abscess be secondary to a chronic middle-ear suppuration, the simpler operation of mastoidectomy if the aural lesion be acute.

In operating upon the mastoid of a patient suspected of harboring a brain abscess, the use of the mallet and chisel should as far as possible be eliminated. This we may usually do altogether in acute mastoid disease by biting off the mastoid tip with a rongeur and continuing the removal of cortex from below upward with the same instrument. In operating upon patients who have suffered from long-standing chronic suppuration, however, the thickness and density of the bone usually require more or less

use of the chisel or gouge. The objection to these instruments depends, of course, upon the possible effect of the resulting concussion upon a collection of pus, perhaps under pressure, within the brain.

The operation upon the mastoid should be performed as quickly as possible. All diseased masses of bone should be dealt with radically. The bony plate forming the roof of the mastoid cavity should be scraped clean of diploic tissue. These essentials having been accomplished, we should waste no time on minor operative details, which may be left to subsequent operation should the need arise. Having exposed the inner plate forming the floor of the mid-cranial cavity, this should be carefully scrutinized for necrotic defects through which pus may be seen escaping from the cranial cavity. Failing to find a fistula leading into the brain cavity, the roof of the mastoid should be removed, and the dural surface thus exposed examined for either a fistulous opening or for pathologic changes indicating a subdural abscess or the subdural terminal point of a fistulous tract leading into the brain substance,—*i.e.*, to a brain abscess.

Treatment of a Brain Abscess Drained through a Dural Fistula.—Should a fistulous opening be found in the dura over the roof of the mastoid, or for that matter in any situation, and pus be seen escaping therefrom, we should consider ourselves fortunate in having discovered a condition which offers a comparatively favorable prognosis. No attempt should be made to enlarge the dural opening. The dura surrounding the fistula should be exposed by the free removal of bone. Fistulous tracts generally do not tend to rapid closure so long as they form the principal pathway for escaping pus. We should not, therefore, attempt to improve upon Nature's provision for drainage,—at least, until it shall have been proved inadequate. The depths of the mastoid wound, and especially the region of the aditus, should be firmly packed with iodoform gauze, while folds of plain sterile gauze should be placed against the dural surface and fistula, and the usual outer mastoid dressing applied.

The dressings should be changed daily. Naturally, any change or recurrence of constitutional or focal symptoms—*e.g.*, temperature or pulse changes, head pain, insomnia, mental obscuration, aphasic symptoms, in short any phenomena which might point to intracranial pus retention—should be looked for and noted.

At the daily changes of dressing, any changes in the condition of the dural fistula should be noted. Our problem here is to maintain the patency of the dural opening. If, therefore, there are physical evidences of its premature closure, we may combat this by the introduction of a small folded gauze wick. What is hoped for, and what probably actually occurs in favorable cases, is the gradual obliteration or shrinkage by cicatricial contraction of the abscess cavity or tract from its depth outward to the dural opening. After this process is completed, there can be no further escape of pus, and spontaneous closure of the dural opening follows.

Naturally, if the surgeon's zeal leads him to persist in introducing drainage tubes or wicks deeply into the fistulous tract, spontaneous healing cannot occur. In a series of ten cases operated upon for brain abscess,

Dean found three with pus escaping through fistulous openings in the dura. In one the dural opening was not enlarged, but a gauze wick was introduced. In the other two the fistulous tracts were absolutely depended upon to maintain drainage, not even the smallest wick being introduced. All of the three cases made perfect recoveries.[5]

Exploration of the Brain.—The mastoid roof having been removed, and no fistulous tract or necrotic area of dura being present, the temporal lobe should be further exposed by removal of a portion of the squama. This may usually be rapidly accomplished by means of a stout rongeur, beginning just above the mastoid and biting away the squama until an area of dura about equal to that shown in Fig. 289 is uncovered. While

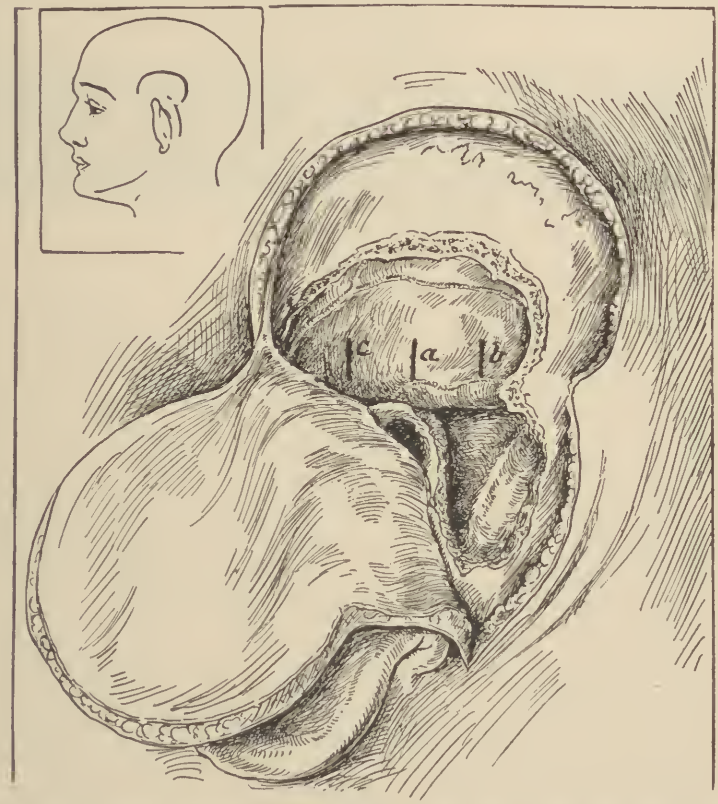

Fig. 289.—Dural exposure preliminary to exploration of the brain for suspected temporosphenoidal abscess.

this may be done by mallet and chisel, it is particularly important to avoid the jar caused by these instruments in the neighborhood of a suspected brain abscess. An area measuring roughly about 1½ inches laterally by 1 inch in height provides a sufficient initial exposure.

[5] Dean, L. W.: Brain Abscess of Otitic Origin, Annals of Otol., vol. xix, No. 3.

Before opening the dura, the whole wound should be flushed with normal saline solution. The mastoid wound and particularly the region of the aditus should be firmly packed with iodoform gauze. The towels about the wound should be changed. A little peroxide of hydrogen may be poured over the exposed dura, which may then be covered with gauze moistened in 1 to 4000 bichloride solution. The surgeon's hánds should be re-sterilized, or, if wearing gloves, these should be changed. All instruments previously used should be removed and only freshly sterilized instruments should be used in the actual exploration of the brain.

For exploring the brain the aspirating needle is not a reliable instrument, as its lumen may become occluded by brain tissue, and, even when this does not occur, it may fail to give passage to thick pus. A more effective instrument is the narrow blade of a long straight bistoury (Fig. 290).

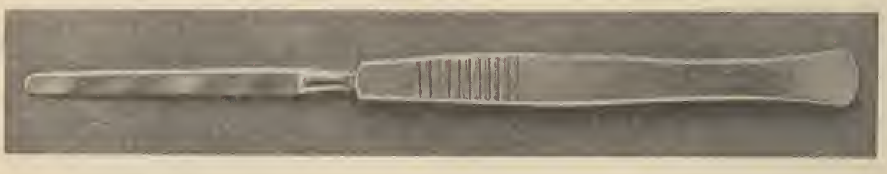

Fig. 290.—Narrow-bladed knife for exploring the brain (size and length reduced).

For one's initial puncture, the logical point of attack is just above the tegmen tympani (Fig. 289, *a*). Holding the knife with the blade surface in the vertical plane,—*i.e.*, with cutting edge directed either upward or downward,—it is introduced directly into the brain substance in a direction at right angles to the anteroposterior plane of the skull. Introduced in this way, the knife makes a short vertical incision through the dura which, whether an abscess is or is not encountered, is not likely to give rise to a cerebral hernia. The knife should enter the brain slowly, its progress being arrested frequently—*i.e.*, at distances of 5 or 6 mm.—and the blade slightly rotated to favor a possible escape of pus. The advantage of this gradual and interrupted introduction must be apparent; for, if we quickly plunge the knife to what we believe to be the limit of safety and an escape of pus follows, we shall be left in doubt as to the depths at which the abscess was reached. If the knife is arrested at a depth of 5 to 6 mm. and pus escapes, we shall be in no doubt as to the superficial character of the lesion, whereas an escape of pus occurring only after it had been introduced to the limit of safety would constitute fairly positive evidence of a deep-seated abscess.

If the knife, introduced, as above described, to a depth of one and a half inches, fails to draw pus, it should be withdrawn and reintroduced in the same careful way at a point about three-eighths of an inch behind the first (*b*). This also failing, the next incision may be made at a point three-eighths of an inch in front of the initial puncture (*c*). It must be remembered that classical focal symptoms of brain abscess have frequently been proved post mortem or by operation to have depended upon very small pus collections; and, further, that, while the most frequent seat of otitic brain abscess is the region in close proximity to the tegmen tympani, such

an abscess may occupy any position within the temporosphenoidal lobe. It may be necessary, therefore, to make a number of closely approximated parallel incisions in order to locate a small abscess giving definite symptoms.

The practice which I have seen advocated, of introducing the knife a second or even a third time through a single dural incision in order to explore the brain in different directions, is clearly inadvisable, for the following reasons: (1) The dural incision itself, provided that perfect asepsis has been maintained, involves little danger; (2) should infection

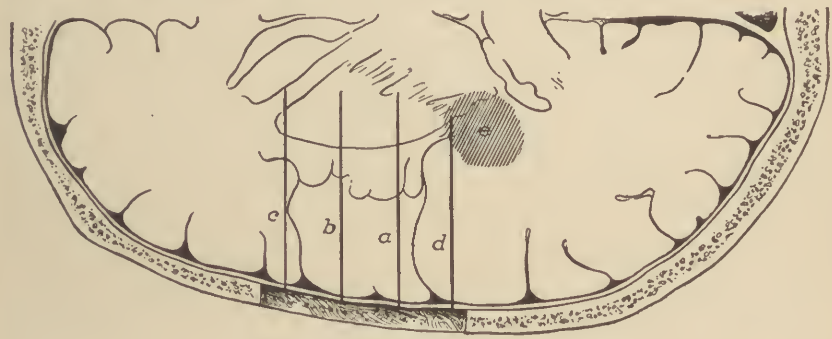

Fig. 291.

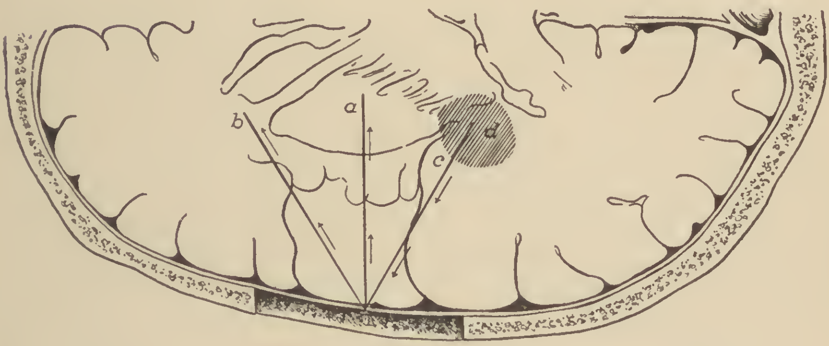

Fig. 292.

Figs. 291 and 292 are drawings made from a section of the brain, and show schematically the advantages of exploring the brain through separate incisions of the dura as compared with exploratory punctures in different directions through a single dural incision. In figure 292 the arrows indicate possible avenues of pus from an evacuated abscess to new foci of infection.

occur later, and pus escape from a dural incision through which the brain had been punctured in different directions, we would be at sea as to the site of the focus of infection; and (3) should the second or third introduction of the knife through a single dural incision reach the abscess sought for, the pus thus evacuated would pass at the dural outlet the open mouths of the tracts made by the first exploratory incisions, and might easily lead to fresh pathways and foci of cerebral infection. This point is made clear by Figs. 291 and 292, in which the possible results of separate incisions and of multiple punctures through a single dural incision are compared.

Surgical experience has abundantly proved that incision of the dura under aseptic precautions does not *per se* involve any very serious danger. Some years ago the writer performed an exploratory operation upon a woman suspected of harboring a brain abscess. Though no abscess was found, the woman made a rapid recovery, without at any time showing symptoms referable to the three incisions through the meninges. A cerebellar abscess operated upon by the writer was evacuated only by the fourth incision, the patient recovering without having exhibited any symptoms traceable to the four dural incisions. A case still more graphically illustrating the tolerance of the meninges to aseptic surgery is that of a brain abscess reported some years ago by Dr. John R. Page.[6] In this case the symptoms pointed strongly to a cerebral abscess, which was finally located and evacuated only after sixteen separate incisions into the brain substance had been made, the patient making a perfect recovery. These cases are cited for the purpose of emphasizing this point,—viz., that, having sufficient grounds for an exploratory operation upon the brain, one should not hesitate to make as many incisions through the meninges and into the brain substance as may be necessary either to locate the abscess or to furnish fairly conclusive evidence of its absence.

The three incisions (*a*, *b*, and *c*, Fig. 289) having failed to locate the abscess, a second row of incisions above those first described should be made. These also failing, the brain in front of and behind the area indicated above should be explored. This may require an even more extensive removal of bone. This, though to be regretted, can not be weighed against the possibility of leaving without external outlet a collection of pus within the brain.

Depth of Exploratory Incisions into the Brain.—Naturally, when a brain abscess is present, the puncture of a ventricle might determine a rapidly fatal result by creating a passage along which the abscess might expel its contents into the ventricle. While I have seen no record of such an accident as a result of an exploratory operation, it is well that such a possibility should be borne in mind. The anterior and posterior horns of the lateral ventricle are in relation respectively to the frontal and occipital lobes rather than to the temporal or parietal. They are on a level from 1¾ to 2 inches above that of the tegmen tympani, and are at least 2½ inches respectively from the upper anterior and upper posterior corners of the dural exposure shown in Fig. 289. In a case of serous meningitis in which from a very similar exposure I intentionally punctured the posterior horn, it seemed to me that the instrument was inserted to a depth of at least 2¾ inches before the volume of escaping liquor cerebri showed that the ventricle had been reached. Why a direct puncture—*i.e.*, horizontally inward—of the temporal lobes from a point about ¾ to 1 inch above the tegmen tympani should not reach the inferior horn of the

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[6] Page: Report of a Case of Brain Abscess, etc., Amer. Journal of Surgery, September, 1908.

lateral ventricle, I do not know. Personally I should feel it necessary to proceed very cautiously at this point after the knife had been carried to a depth of 1¼ inches. Heine, in discussing the safety limit as to the depth of exploratory punctures, quotes Körner as placing 4 cm. (about 1⅝ inches) as the limit of safety. Heine, however, cites a case operated upon by himself in which an abscess was reached only at a depth of 7 cm. (about 2⅝ inches) from the exposed dural surface, and states his belief that, in the nature of the lesion, it is essential that one be not too closely bound by dogmatic rules as to the position of important structures and their relation expressed in millimetres to the cerebral cortex.

Drainage.—A brain abscess having been opened, whatever subsequent treatment will maintain the patency of the drainage pathway and at the same time subject both brain and meninges to minimum disturbance and manipulation, will in the long run secure the best average results.

Irrigation of the abscess cavity, while it has been successfully used in certain cases, is a measure not of itself without possibilities of harm, and is no longer advocated.

The practice of making a crucial incision of the dura is not essential to adequate drainage. By weakening the dural support, it may pave the way for a small hernia cerebri, and to this extent certainly increases the risk of subsequent meningeal infection.

On no account should the finger be introduced into the freshly made tract by which the abscess is being drained.

If the surgeon is convinced of the necessity of mechanically aiding in the removal of pus, Whiting's encephaloscope (Fig. 293) may be carefully introduced, and pus as it flows into the lumen of this speculum may be wiped out by means of cotton-tipped applicators. But to feel that one is called upon to explore the nooks and corners of the abscess cavity by means of this or any other instrument is likely to lead to the infliction of mechanical injuries, possibly giving rise to fresh pathways or foci of infection.

Having created an avenue of escape,—presumably a path of least resistance,—the most important agency in maintaining adequate drainage is the evenly distributed force of normal intracranial pressure. Suppose, for example, in a given case that diverticula containing pus do exist. Presumably they were caused by pus under pressure seeking escape. Pressure within the abscess cavity having been relieved by a newly made pathway of escape, it seems not illogical to expect upon purely physical grounds that small communicating spaces containing pus may drain back into the main abscess cavity and not refill so long as the outlet of escape is maintained. I believe, therefore, that we should not attempt to explore by actual touch or sight the brain abscess which we have just succeeded in opening.

When pus following the knife shows that the abscess has been located, it should be held steadily in place and slightly rotated until pus ceases to flow. It may then be used as a guide for the introduction of whatever instrument is to take its place or for the permanent wick or drain.

If the abscess is deep-seated, Heine advises that drainage be maintained by a piece of perforated rubber tubing, wrapped in iodoform gauze and introduced not too deeply into the abscess cavity. Theoretically such a drain would seem to the writer to involve considerable risks in the

Fig. 293.—Whiting's encephaloscope.

mechanical irritation to which it might give rise. The use of absorbable decalcified chicken bone, which was employed so successfully by Macewen, is open to certain obvious objections, and is not in general favor to-day. McKernon's choice—*i.e.*, of a cigarette gauze wick rolled in a mixture of

equal parts of powdered boric acid and iodoform—gave excellent results in a series of cases operated upon by him.

As to the problem of keeping the drain or wick in proper position, the writer learned a simple expedient from the following probably fairly common experience. In making the first change of dressings in an abscess case, the gauze wick was found lying flat upon the dural surface, having been expelled by intracranial pressure. To ensure against a repetition of this mishap, he had a number of wicks prepared in the following way: First, a small flat pad of three or four folds of gauze was made. With the pointed blade of a scissors a small hole was made in the centre of this pad, and through this a loop of infolded gauze wick was forced (Fig. 294). A number of these were prepared and sterilized, each in a separate wrapper. In actual use they seemed to have the following advantages: (1) Having determined the depth to which we wish the wick to penetrate the brain,—say one inch or an inch and a quarter,—the loop of gauze wick is forced through its fixating pad to this length. When this loop-wick has been introduced into the abscess cavity, the pad is flattened against the surrounding dural surface and sterile gauze is packed around it. In this way the wick is held immovably in place and by a means which in no way interferes with drainage and cannot possibly produce irritation of surrounding tissues. (2) Having thus to arrange in advance the length of each wick introduced, the surgeon is never in doubt as to the depth to which the wound is being packed, and he is in a position to arrange systematically for a gradual and progressive shortening of the wick.

Fig. 294.—Gauze wick for use in brain abscess. Only the loop (*a*) is introduced into the abscess tract and cannot, therefore, leave threads behind it on withdrawal. The length can be regulated at will, and when gauze is packed around the fixation pad, the wick cannot be displaced or expelled by intracranial pressure.

Many surgeons prefer to provide drainage in brain abscess cases by means of a small flattened roll of sterilized rubber tissue, claiming an advantage in the smoother surface as less likely to irritate contiguous brain tissue.

Personally, I believe that the dressings in brain abscess cases should be changed daily, the saturated wicks being removed and dry sterile ones replaced with the least possible friction or mechanical disturbance of the tissues involved.

Should an occasional escape of pus seem in excess of that usually coming away with the change of dressings, this of itself should not be regarded as an indication for further exploration or subdural manipulation of the

brain. Having established drainage, and the symptoms of intracranial disturbance having subsided, I believe that nothing short of a recurrence of symptoms pointing to pus retention should tempt the surgeon to a further exploration of the interior of the brain.

Finally, it is of the greatest importance that the patient—however favorably he may appear to be progressing toward recovery—should for a considerable period be kept absolutely quiet in bed. The surgeon should be in no hurry to close the wound. The two chief dangers of the postoperative period—viz., secondary retention of pus within the brain and late infection of the meninges—are always present during the first weeks following the operation. Until the period of postoperative danger is definitely passed, the patient should be kept quiet in bed and guarded against any indiscretion which might turn the scale unfavorably against recovery.

Cerebellar Abscess. — The surgical treatment of cerebellar abscess is practically the same as that of abscess of the temporal lobe. Since an epidural abscess if present is most likely to be found in close relation to the posterior surface of the petrous bone, the cerebellar dura should first be exposed in this situation.

This is best done by uncovering first the sigmoid sinus, and then with a suitable rongeur removing the bone in front of and internal to its anterior border. When sufficient bone has been removed, we have an exposure of cerebellar dura throughout a triangular space bounded above by the tegmen antri, behind by the anterior margin at the sigmoid sinus, and in front by the petrous bone (Fig. 297, *a*).

Should a fistulous opening in the dura be present in this area, it should be treated by exactly the same conservative method as that already advised in the case of temporal lobe abscesses drained through pre-existing dural fistulæ.

As in the case of temporal lobe abscess, the dura, though intact, may show pathologic changes indicating the site of a subdural collection of pus or the subdural terminal point of a necrotic tract leading to a cerebellar abscess.

Even though no such pathologic changes are present, an effort may be made to locate the abscess through the dural exposure in front of the sinus. Before incising the dura, the wound should be cleansed, the aditus firmly packed with iodoform gauze, fresh towels placed about the wound, and the surgeon's hands re-sterilized.

In the first incision the knife is carried directly inward to a depth of not more than one inch (Fig. 295, *a*). This failing, a second incision, made through a separate dural opening behind the first, is carried inward and slightly backward to a depth of 1⅛,—not more than 1¼ inches (*b*). Should this also not give exit to pus, it is better that no further effort be made to locate the abscess from this exposure. The mastoid wound is now packed with iodoform gauze, plain sterile gauze, however, being placed against the incised dura.

It is now necessary to expose the cerebellum behind the sigmoid sinus by a removal of bone from that part of the occipital bone forming the outer wall of the cerebellar fossa. This may be done by trephining at a point one inch behind the centre of the sigmoid sinus and well below the level of the lateral sinus; or, better, by means of a stout rongeur we may continue the removal of bone directly backward from the posterior border of the sigmoid sinus. The results of the two methods of exposing the occipital aspect of the cerebellum are shown in Figs. 296 and 297. In these illustrations the sigmoid sinus is shown somewhat diagrammatically. That is to say, the bony support having been removed, the rounded contour of the sinus is usually lost, and the line of demarcation between the dura covering the sinus and that covering the cerebellum is, therefore, not so clearly defined as appears in the illustrations.

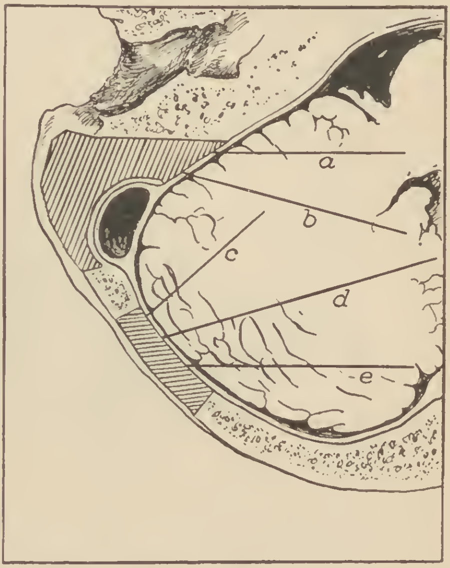

FIG. 295.

The posterolateral aspect of the cerebellum having been exposed, we should adopt some systematic plan of completing the search for the suspected abscess. As with the temporal lobe, each exploratory puncture should be made through a separate dural incision. The first, incising the dura well forward, enters the cerebellum in a direction inward and strongly forward, and to a depth of one inch (Fig. 295, *c*). The next incision, cutting the dura a little behind the first opening, passes inward and slightly forward to a depth of 1½ inches (*d*). This also failing to draw pus, a fifth incision (*e*) is carried directly inward through the posterior part of the cerebellum to a depth of 1⅛ to 1¼ inches.

The writer some years ago performed an exploratory operation upon the cerebellum which failed to locate an abscess. The autopsy, made by Dr. Zabriskie, showed a small deep-seated abscess to have been present, and that one incision, which traversed the cerebellum in the right direction, had failed by an eighth of an inch to enter the abscess cavity. Realizing that this failure was in some degree due to a lack of adequate knowledge of cerebellar measurements, the writer has had forcibly impressed upon him the importance of systematic method and thoroughness in an operation of this nature. Fig. 295 represents an effort to place at the student's disposal a chart giving approximately the directions and depths

of exploratory punctures of the cerebellum which should fairly well cover the region to be explored. If these several efforts fail to locate the abscess, and the surgeon is still uncertain as to its possible presence above or below the plane of the incision herein described, one or more supplementary incisions above and below may be made.

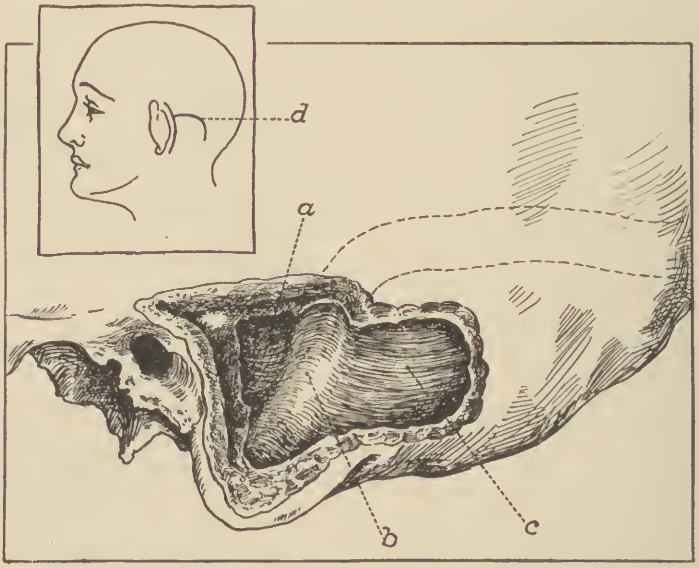

Fig. 296.—Exposure of dura covering cerebellum: *d*, supplementary skin incision for adequate exposure of bone; *a*, cerebellar region in front of sigmoid sinus; *b*, sigmoid sinus; *c*, cerebellar region behind the sigmoid sinus.

The abscess having been located, the subsequent drainage and the after-treatment may be carried out by practically the same method as has been described in connection with abscess of the temporal lobe.

Meningitis.—The surgical treatment of otitic meningitis has already been discussed in connection with the pathologic and clinical aspects of the various recognized types of the disease. All that is called for here, therefore, is a very brief *résumé* of the different surgical procedures which have proved their value in certain cases, and of the not too clearly defined indications for their use.

Serous Meningitis: *Treatment.*—All that may be necessary is a very careful and rather wide uncovering of the dural area involved. Particularly in children are symptoms of marked meningeal irritation frequently relieved by this comparatively simple operation. Unless the symptoms or the pathologic changes in the bone suggest the posterior fossa as the probable seat of the meningeal lesion, the dura covering the temporal lobe should be first exposed. This may be done in exactly the same way as the preliminary exposure for exploring the brain in a case of suspected temporal lobe

abscess,—*i.e.*, the tegmen tympani et antri is first removed and then the adjacent portion of the superior plate of the squama. The writer recalls the case of a child who, following a simple mastoid operation, developed high fever, continuous at 104° F. or thereabout, rigidity of the neck muscles, delirium, and strabismus, all of which disappeared within a few days after the operation referred to above.

Should the surgeon have reason to fear that the cerebellar dura is the seat of trouble, this region also should be exposed by a removal of bone just in front of the sigmoid sinus (see Fig. 297).

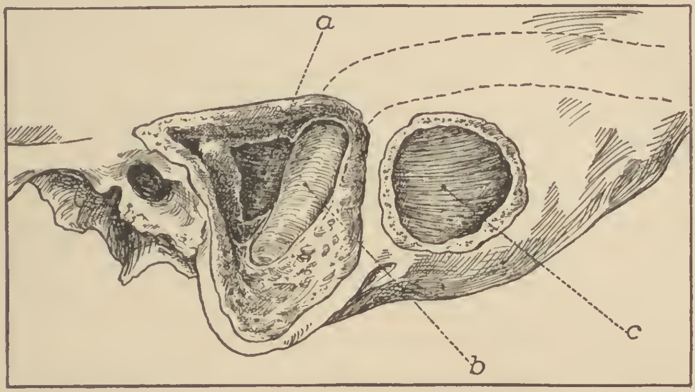

FIG. 297.—Exposure of dura covering cerebellum: *a*, cerebellar region in front of sigmoid sinus; *b*, sigmoid sinus; *c*, cerebellar region behind sigmoid sinus.

Simple uncovering of the dura is particularly indicated in the case of young children, who are more prone than adults to the development of mild forms of meningeal inflammation, and whose symptoms are also likely to be far more pronounced. I believe also that it should be used as a preliminary measure in all comparatively mild cases, whether occurring in children or adults, in which lumbar puncture fails to reveal pus or bacteria in the spinal fluid. If successful, there is usually some amelioration of symptoms or improvement in the patient's general condition within twenty-four hours. If no improvement is realized, the operation may then be quickly and easily supplemented by one or more incisions of the dura.

The above statement refers to cases of serous meningitis of moderate or average severity. Undoubtedly there are cases in which the symptoms assume early so alarming a type that one is impelled at once to adopt more radical measures. When, following symptoms of meningeal irritation, the patient quickly lapses into delirium and shows the peculiar depression of the vital forces characteristic of grave meningeal disturbance, there should be no loss of time in relieving tension by several incisions through the dura.

Puncture of the Lateral Ventricle.—Withdrawal of fluid by lumbar puncture has been advised as a means of obtaining prompt relief of pres-

sure in severe cases. The exact indications for this procedure have not yet been determined. The writer has had under his care one apparently hopeless case of serous meningitis (see pages 390–391) in which tapping of the ventricle was followed by almost spectacular relief of symptoms. While the result in this case was most gratifying, there has always existed a doubt in the writer's mind as to whether the ultimate result might not have been the same had he limited the extent of his intervention to the three vertical incisions of the dura which formed part of the operation.

Serous Meningo-encephalitis (Körner). — This condition, probably analogous in its origin and inception with serous meningitis, differs chiefly in the extension of the morbid changes into the substance of the cerebral cortex. In some cases the outer brain structures bear the brunt of the attack, the symptoms suggesting a cerebral rather than a meningeal lesion. Such cases have been reported by Jansen, Arnold Knapp, the writer, and others (see cases cited in Chapter XIII).

The surgical treatment of otitic serous encephalitis begins by exposure of the dura covering the temporosphenoidal lobe as for an exploratory operation for brain abscess (Fig. 289). Under the strictest aseptic precautions, two or three parallel vertical incisions through the dura, and passing to a depth of a half inch directly into the brain substance, are made. No drains should be introduced for the purpose of maintaining the patency of these openings. Rather loose folds of sterile gauze are placed against the incised dura. In favorable cases the improvement in the patient's condition is soon noticeable, and the symptoms, once relieved, do not, as a rule, recur. Following this operation there is likely to be a rather copious leakage of cerebrospinal fluid into the dressings, which may persist, but in gradually diminishing amount, for two or three weeks.

Should the meningeal symptoms, having once definitely subsided, recur, their reappearance will usually be found to depend upon the development of diffuse suppurative leptomeningitis. The importance of maintaining the most careful asepsis during the postoperative period must, therefore, be apparent.

Purulent Leptomeningitis. — The surgical treatment of purulent leptomeningitis, circumscribed or diffuse, may be dealt with briefly. The dura covering the under and lower-lateral surface of the temporosphenoidal lobe should be exposed; and unless there is physical evidence that the disease is located there, the cerebellar dura should also be uncovered by the removal of bone in front of and internal to the sigmoid sinus. The exposed dura in both regions should be closely scrutinized for physical evidences of disease. When inflammatory changes confined to a limited area point with probability to the existence of a circumscribed leptomeningitis, the area involved should be freely incised in parallel lines. Unless the meningeal lesion is complicated by a subdural collection of pus of appreciable size, drainage wicks should not be introduced. Sterile gauze in loose folds is placed against the dural incisions, and a large absorbent dressing applied. The dressing should be changed daily, the frequent

removal of surface pus and pus-soaked gauze being necessary to promote drainage. When it is deemed necessary to maintain mechanically the patency of the dural openings, the lips of the incisions may be separated occasionally by means of a grooved director or other blunt instrument. As a rule, this is not necessary.

When the macroscopic changes in the dura and the symptoms both point to the presence of a diffuse or spreading suppurative leptomeningitis, the lesion, so far as our present knowledge goes, is a hopeless one. Usually the character of the changes is such as will not be relieved by drainage, and furthermore the area involved is too extensive to be reached by any operative procedure. While the prognosis is, therefore, most unfavorable, I believe in giving the patient the benefit of any theoretic chance. The dura of the temporosphenoidal lobe should, therefore, be freely drained by multiple incisions. Regarding the disease as having some analogy with other suppurative lesions, the above treatment is at least in accord with surgical principles. The postoperative treatment is the same as for circumscribed purulent meningitis,—daily very careful renewal of the dressings.

The puncturing of the lateral ventricle in diffuse purulent leptomeningitis does not appeal to me as a rational or promising measure.

CHAPTER XIX.

FACIAL PARALYSIS.

Facial paralysis of otitic origin may result from any of the following conditions: (1) Acute suppurative otitis media of severe type, the inflammatory process extending through the tympanic wall of the Fallopian canal, and the nerve being either directly involved or subjected to pressure by inflammatory products within the canal. This is commoner in young children than in adults. (2) During acute suppurative otitis media as a result of direct exposure of the nerve through a defect in the tympanic wall of the facial canal. (3) In chronic suppurative otitis media, the necrotic process involving the facial canal. (4) In suppurative labyrinthitis secondary to chronic middle-ear suppuration, a necrotic tract through the horizontal semicircular canal may by downward extension involve the facial. (5) Facial paralysis is an occasional accompaniment of otitic meningitis, in which case the nerve lesion is probably more often the result of an intermediate infection of the labyrinth than of an extension of inflammation from the meninges to the nerve-trunk as it traverses the internal auditory canal. (6) Tuberculous lesions. In a very large precentage of cases of middle-ear tuberculosis, the facial nerve is involved. A tubercular element in the pathogenesis of facial paralysis is recognized as affecting the prognosis unfavorably, from the greater frequency with which the nerve is actually destroyed. (7) Herpes zoster auriculæ (Hunt) is an occasional cause of facial paralysis. In this affection the morbid changes in the nerve are clearly an extension of the inflammatory process primarily affecting the geniculate ganglion.

Generally speaking, facial paralysis resulting directly from middle-ear or mastoid suppuration—the labyrinth having escaped infection—offers, under prompt and rational treatment, a prefectly favorable prognosis. Usually all that is required to effect a cure is careful removal of all diseased bone through a simple mastoidectomy or radical cperation, according to the nature of the tympanic lesion.

Postoperative facial paralysis may occur: (a) as the immediate result of division or injury of the nerve during a radical operation: or (b) as the deferred result of a slight traumatism, the paralysis appearing only after an interval of several hours or days has elapsed. (c) When the tympanic wall of the facial canal is either defective or unusually thin, paralysis may result directly from the pressure of a gauze dressing packed too tightly into the wound cavity. (d) A thick-walled, sclerotic mastoid with a very small, deeply-placed antrum is a type of bone in which during a simple mastoidectomy great care may be necessary to avoid injury to the nerve. The danger in this type of bone lies in the fact that the uniform or average density of bone is such as to obscure certain common surgical landmarks, and in the

search for a small antrum—often hardly extending beyond the usual confines of the aditus—the nerve is injured before the surgeon recognizes the surgical region he has reached. A particularly distressing feature of an accident to the nerve occurring in this way is the fact that the injury is likely to be complete, and the resulting paralysis therefore permanent. The deduction from this statement is practical and clear: *i.e.*, that the surgeon, having recognized the type of bone he has to deal with, should either proceed with unusual and infinite care, or make use of the postero-superior wall of the bony auditory canal as a guide to the aditus and antrum. It is better to sacrifice any non-vital structure than permanently injure the facial nerve.

The regions in which the nerve is most likely to be injured were spoken of in connection with the technic of the radical mastoid operation (page 442), and need not be referred to here.

Symptoms and Physical Signs.—The facial changes accompanying this lesion are so characteristic and constitute a deformity so pathognomonic as to overbalance in diagnostic importance all other clinical features.

Owing to the paralysis of the facial muscles corresponding to the side of the aural lesion, the normal facial folds are obliterated. Thus the wrinkles or lines of the forehead and about the eye and the nasolabial fold disappear wholly. The skin, therefore, presents a smooth and lineless surface in conspicuous contrast to the opposite side of the face. In children and young adults this may not be conspicuous while the face is in repose, but becomes unpleasantly noticeable as soon as the sound facial muscles are called into activity,—*e.g.*, in laughing, crying, etc. The patient cannot close the eye on the affected side, and cannot contract the labial muscles. In cases of complete paralysis, the affected side of the mouth may droop to such an extent as to allow a more or less constant escape of saliva.

As a result of the physical changes above noted, the patient may experience the following disturbances of the special senses: The inability properly to close the eyelids leaves the eye unprotected, and the conjunctiva and even the cornea may become inflamed or congested. Owing to paralysis of the nasal muscles and collapse or flaccidity of the ala nasi, the passage of air to the upper nasal chamber is diminished or prevented, and to this extent the olfactory sense is disturbed. Paralysis of the chorda tympani nerve is responsible for loss or disturbance of taste in the corresponding half of the tongue. Finally, paralysis of the stapedius may result in a very annoying disturbance of the auditory function, which takes the form of a loss of auditory balance or power of accommodation to various sounds rather than of an actual diminution of hearing. In many cases the paralysis of the stapedius in persons whose hearing has previously not shown great impairment is said to result in an appreciable hyperacusis for the lower musical tones.

The recognition of these various disturbances of the special senses is said in some cases to be of value in determining the location of the surgical injury to the nerve. Thus, division of the nerve within the facial canal at

a point between its exit at the stylomastoid foramen and the point at which the nerve to the stapedius is given off, would involve the chorda tympani and cause disturbances of taste, but would not give rise to any disturbance of the auditory function. Division of the nerve above the branch to the stapedius would add to those just mentioned the symptoms of disturbed audition,—*i.e.*, some confusion of sound impressions, hyperacusis for the lower tones, tinnitus aurium, etc.

When the paralysis is complete and attended by characteristic electrical reactions of degeneration, marked atrophy of the paralyzed facial muscles may add to the disfigurement.

Prognosis.—The prognosis depends upon the character of the injury. Actual division, unless the segments are maintained in apposition or are subsequently reunited, means permanent paralysis. When the nerve is not actually divided, recovery, partial or complete, occurs in the great majority of cases. As to the duration of the paralysis in any particular case, but little can be said. A paralysis due to pressure may recover completely within a few weeks, whereas a lesion due to a severe traumatism, giving rise to complete degeneration of the peripheral segment, may cause a paralysis lasting a year or more and yet result ultimately in complete recovery.

Treatment.—When there is no reason to fear that the nerve has been actually divided, the patient should be told that ultimate recovery is to be expected. The greatest care should be given to keeping the wound clean and free of pus. Tight packing of the wound cavity should be avoided. The facial muscles should be regularly massaged to forestall or combat muscular atrophy. For the same purpose the faradic current should be used daily or at least on alternate days. Strychnine sulphate, in doses of gr. 1/60 three times a day, and continued over a considerable period, is advised, but whether it actually influences the lesion is open to question. These measures should be persisted in, and it is usually necessary gradually to acquaint the patient with the fact that several months may elapse before the earliest signs of returning function will be noticeable.

Before leaving the subject, a word should be said as to the duty of the surgeon who is obliged to face the responsibility of a case upon which he has operated and in which he is convinced that he has actually divided the nerve. In the first place, should the nerve be divided during an operation and the accident be recognized at the time of its occurrence, there would seem to be every reason for attempting at once or within a few days of the injury to reunite the segments within the tympanic cavity. Presumably in most cases the injury occurs at some distance from the geniculate ganglion, and I can see no reason why the Fallopian canal should not be carefully followed and opened in both directions,—*i.e.*, backward toward the bend and then along the descending portion,—and also in the opposite direction,—*i.e.*, forward toward its point of entrance into the tympanic cavity. With bright illumination, an enlarged radical cavity, and with patient, careful work, I can see no reason why it should not be

perfectly practicable to take up the divided segments of the nerve within the tympanic cavity and reunite them with a suture of fine silk.

When the knowledge that the nerve has been actually divided or permanently injured comes to the surgeon only through the lapse of time and absence of any signs of returning function, the question of re-establishing function by joining the degenerated peripheral end of the facial to an intact cranial nerve (hypoglossal or spinal accessory) must be considered.

While the results of such attempts in the case of the facial nerve have been by no means uniformly successful yet I am of the opinion that, when recovery is otherwise hopeless, the operation should be undertaken, with the view to obtaining as much restoration of function as possible, and, even should this prove disappointing, to securing sufficient regeneration to prevent extreme muscular atrophy.

Of the two cranial nerves usually considered,—*i.e.*, the hypoglossal and the spinal accessory,—the former is the more favorable, for the following reasons: The spinal accessory is the motor nerve of the sterno-mastoid and trapezius muscles; when a successful anastomosis between it and the peripheral segment of the facial has been accomplished, voluntary movements of the shoulder are accompanied by involuntary facial twitching, while voluntary movements of the face are associated with involuntary shoulder movements.

The writer believes that, from the viewpoint of safety, the operation for facio-hypoglossal anastomosis belongs neither to the aural surgeon nor the average general surgeon. Rather it should be entrusted to the man in any community or section who by animal experimentation, special anatomical study, dissection work, etc., and by the average results of such operations as have come to him, has established his special fitness for this peculiarly delicate work. On the other hand, conditions are easily conceivable—*e.g.*, in regions remote from the large medical centres—in which such cases must be operated upon by competent surgeons without this special training, or not at all. We, therefore, include a brief outline of the surgical steps for uniting the peripheral segment of the facial to the trunk of the hypoglossal.

The incision for exposing the facial nerve begins at the anterior border of the mastoid process and is continued along the anterior margin of the sterno-mastoid muscle to a length of about two inches. This incision, which successively divides all the tissues down to the deep cervical fascia, should be made with the greatest care not to injure the parotid gland. When the deep facia has been reached, a finger introduced into the depth of the wound will easily locate in its upper part and in a position corresponding to a point just in front of the mastoid tip, a surgical landmark of practical importance, *i.e.*, the styloid process. Around this process the facial trunk turns and in this situation it can be felt as a cord-like structure rolling between the process and finger (Taylor). From this point, it passes forward to enter the parotid. It is infinitely safer to locate the nerve thus as it crosses the styloid process than to look for it at its point of entrance into the parotid canal. Having determined by palpation its position and direction, it

should be exposed by dividing the overlying tissues in a direction parallel to its course. To attempt to locate it by the sense of sight through a gradually deepening incision running cross-wise to the nerve trunk is almost certainly to divide it. Once exposed and freed, it should be traced backward and then upward toward the stylo-mastoid foramen. The mastoid tip, if present and obstructing the way, should be removed. The nerve is then followed to its point of exit from the skull, and divided at the highest point within the stylo-mastoid foramen that a small, sharp,angular knife will reach.

The second step of the operation is the more difficult one of isolating the hypoglossal nerve. The first surgical landmark is located by palpation,—as a smooth prominence representing the transverse process of the atlas. Two structures to be looked for in order to avoid their injury are the occipital artery and the spinal accessory nerve. The artery is occasionally encountered passing upward and outward across the transverse process of the atlas, and should be either retracted or divided between ligatures in the interest of a clear surgical field. The spinal accessory, also passing over the same process, sometimes in front of and sometimes behind the internal jugular vein, should also be borne in mind and retracted to a position of safety. Both of these structures, as also the internal jugular vein, are covered by a layer of deep fascia, which should be carefully divided by an incision beginning over the transverse process, and then extended in both directions to a total length of about 1½ inches. This incision exposes the internal jugular vein, which should be carefully freed by blunt dissection posteriorly to permit of its being gently retracted forward. This retraction is mechanically equivalent to its rotation forward and should bring into view the two characteristic cord-like structures of the hypoglossal and vagus nerves, which in their normal relation in this location lie between and rather behind the internal jugular vein and internal carotid artery. Usually the hypoglossal is the more superficial of the two. It may be identified by mechanical stimulation which should cause twitching or contraction of its appropriate muscles (styloglossus, hyoglossus, geniohyoid, geniohyoglossus, thyrohyoid, sternothyroid, sternohyoid), or, perhaps better, it may be traced downward to the point where it gives off the descendens noni, and bends forward, crossing the occipital and external carotid arteries to pass to its distribution in the tongue. Having isolated and surely identified the nerve, it should be divided, if an end-to-end anastomosis is contemplated, at a sufficiently distal point to allow of its easy approximation—*i.e.*, without tension—to the peripheral segment of the facial. The nerves are held together in end-to-end apposition by sutures of fine silk passing only through their neural sheaths. The wound surfaces are allowed to come together and sutured.

Dr. Alfred S. Taylor,[1] whose paper on the surgical treatment of facial paralysis was published as long ago as 1904, and who possibly has operated upon more of these cases than any other man in New York, prefers not to

[1] Taylor: Surgical treatment of the facial palsy, with technique or facio-hypoglossal anastomosis: Medical Record, February 27, 1904.

Fig. 298.—Exposure of facial and hypoglossal nerves (Taylor). (1) Styloid process; (2) facial nerve; (3) hypoglossal nerve; (4) vagus.

Fig. 299.—Lateral implantation of facial segment intohypoglossal trunk (Taylor). (1) Styloid process; (2) facial nerve; (3) hypoglossal nerve; (4) vagus.

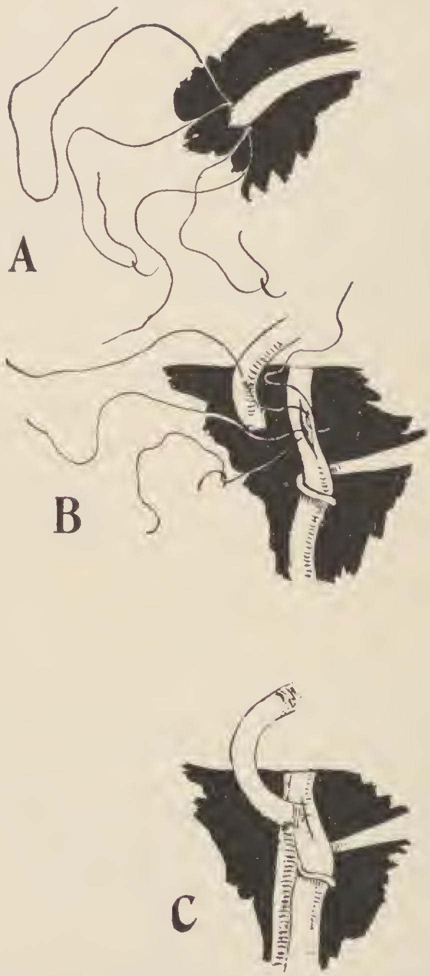

Fig. 300.—Anastomosis of facial and hypoglossal nerves. A, Distal segment of facial nerve. B, Longitudinal slit in trunk of hypoglossal. C, Implantation of facial in hypoglossal trunk (Taylor).

join the facial and hypoglossal nerves in end-to-end anastomosis, but rather to implant the end of the divided facial laterally into a longitudinal slit in the hypoglossal nerve trunk. The advantage claimed for this operation is the fact that paralysis and atrophy of the muscles supplied by the hypoglossal are obviated.

There has, I believe, been a very considerable diversity of opinion as to the relative value of end-to-end anastomosis of two divided nerves, and lateral implantation. Certain authors of repute have gone so far as to state that lateral implantation should never be employed. Such sweeping statements and condemnations are unfortunate in the errors to which they occasionally give rise. The only valid argument which could be brought against lateral implantation in the case of the facial and hypoglassal nerves would be the fact that in actual experience the desired regenerative processes were not induced; but since the average results have been excellent, this argument does not here apply. Surely, unless some very definite gain can be assured, the actual division of any of the cranial nerves should be avoided.

I am indebted to Doctor Taylor for permission to use the accompanying illustrations (Figs. 298, 299 and 300).

Those who have witnessed his technic and the comparative ease and freedom from avoidable traumatism which his skill and experience bring to this work, will the more readily agree with my contention that these cases should be entrusted only to those whose special training best fits them to accomplish the mechanical and physiological results which are the ends in view.

Even when the mechanical work of this operation has been successfully executed, both surgeon and patient must be prepared for a considerable lapse of time before any evidences of returning function will be noticeable. Bowlby,[2] than whom perhaps no man has had a larger experience in the surgical treatment of injured and degenerated nerves, has this to say in regard to the interval which in cases of long-standing degeneration must be allowed for between the neurorrhaphy and the reëstablishment of function: "What most interested me was to find that in many patients who had completely recovered, there had been a long period of many months, or even years, between the time of injury and the return of sensation or motion, and I saw that no case should be written down as a failure simply because the operation of suture was not followed by immediate success. . . . If there is one fact more than another which stands out in the clinical histories of the patients who have been under my own observation, it is that, after the failure of union by first intention, after trophic changes of many kinds, after complete atrophy and degeneration of the paralyzed muscles, recovery may yet be complete."

[2] Bowlby, Anthony A.: Injuries and Diseases of Nerves and their Surgical Treatment, Blakiston, Phila., p. 155.

CHAPTER XX.

CONGENITAL ABNORMALITIES OF THE AURICLE.

The anatomical defects, or abnormalities, of the auricle are so varied as almost to defy concise classification. Within certain limits, considerable departures from the normal standard either as to size or shape may attract comparatively little notice so long as the two ears are alike. On the other hand, a very slight unilateral abnormality may constitute a noticeable deformity. For example, few simple defects attract the gaze more insistently than asymmetry in position,—*e.g.*, one auricle standing more prominently from the side of the head than its fellow. Again, different parts of the auricle vary in the extent to which any anatomical variation engages the eye. Thus, very considerable changes in the arrangement of the cartilaginous folds may pass unnoticed, while even moderate variation in the size or shape of the lobule will attract the gaze unpleasantly and must therefore be considered a deformity. As to the variations in size—the auricles being normal in contour and position—a small ear is usually accounted beautiful, while any enlargement above the normal standard is unattractive and therefore popularly accounted a defect.

Among the recognized types of aural deformity may be mentioned the following:

I. Macrotia, a term used to describe anatomical conditions resulting in abnormal increase in the size of the auricle. The increase in size may be due to uniform or symmetrical enlargement of all its parts or, as is more frequently the case, may be due to redundancy of that portion of the auricular cartilaginous plate situated between the margin of the concha and the margin of the helix. If, to offset this, the peripheral margin is deeply infolded, the general outline of the ear may not be greatly enlarged, but there is produced the disfiguring anomaly described as the "redundant helix" (Fig. 301). When this deep infolding of the helix does not occur, the prolongation of the auricular plate—even though it be extended in the normal plane—increases noticeably the distance between the peripheral margin, or helix, and the side of the head, giving the effect of abnormal protrusion (Fig. 302). When the upper part of the auricular plate is narrowed and prolonged upward so as to represent a point instead of the usual curve, the peculiar type known as a "satyr's ear" is produced (Fig. 303).

Redundancy of the lobule may form part of a uniform enlargement or may occur independently. It produces a conspicuous abnormality, which, fortunately, is easily corrected.

II. Polyotia, or the occurrence of supernumerary auricles, is, fortunately, a rare condition, of which, however, authentic cases have been recorded.

III. Supernumerary Parts.—Of much more frequent occurrence is the condition in which there are supernumerary parts, usually small

cartilaginous nodules covered with normal skin and closely appended to the auricle. Their most usual position is immediately in front of or below the tragus, or below the inferior auricular fold,—*i.e.*, beneath and below

FIG. 301. FIG. 302. FIG. 303.

FIGS. 301, 302, 303.—Three common types of macrotia.

FIG. 304.—Supernumerary cartilaginous nodules.

FIG. 305.—Supernumerary nodules.

the lobule. This congenital abnormality is not very unusual,—several cases being seen yearly in any large clinic,—and is usually associated with an otherwise normal auricle (Figs. 304 and 305).

IV. Projecting Auricle, "Lop Ear" (Fig. 306).—This condition is usually symmetrical,—*i.e.*, involves equally the two ears. In some cases it is apparently due to a redundancy of the entire cartilaginous plate from the margins of the cartilaginous meatus upward and backward to the helix. As a consequence, the cartilage is curled outward or forward as well as enlarged. Occasionally with this condition there is in one or both ears an obliteration of the normal bend or fold of the antihelix, which increases greatly the deformity by throwing the auricle still further forward and downward. In another class of projecting ears the displacement is apparently due to a deepening of the cavity of the concha, this giving rise to a prominent hemispherical projection on the posterior surface of the auricle which throws the ear forward.

Fig. 306.—Projecting auricle; "lop ear."

In all the above conditions the ears may be and usually are functionally normal, or at least there is no functional impairment dependent upon the auricular abnormality. The treatment is, therefore, undertaken solely for the purpose of relieving deformity or improving personal appearance. This, however, is no reason for regarding this field of work as not worthy of special study.

V. Microtia.—A class of deformities coming under a wholly different category from any of the above are those grouped under the general term microtia. Meaning literally abnormal reduction of the size of the ear, it usually describes anomalies of form so gross that all resemblance to the normal auricle is lost. Moreover, it generally includes malformation or absence of the auditory canal, and frequently changes in the tympanic or even in the labyrinthine structures. Microtia is usually associated, therefore, with serious loss of hearing. In some cases not only the membrano-cartilaginous meatus but also the osseous meatus is completely absent. As microtia is very often bilateral, many of its unfortunate victims are practically deaf-mutes. As to the auricular deformity, it includes a variety of conditions from a shrivelled and misshaped roll of cartilage to a keloid-like band of skin-covered cartilage with complete absence of auditory meatus (Figs. 307 and 308).

From the viewpoint of the deformity, the possibilities of treatment may be dealt with briefly. In a majority of cases the auricular cartilage is so rudimentary or the malformation so gross that by no wizardry of plastic surgery could any semblance to a normal auricle be produced. But there is

a more important side to the question of treatment,—*i.e.*, the possibility of surgical measures for the improvement or more often the establishment of hearing. Any discussion of this question must take the form of a discussion of the various observations and theories as to the morbid changes usually present. Does a grossly malformed auricle with deafnesss usually or always indicate a coincident defect of the auditory labyrinth? Does congenital atresia of the auditory canal necessarily imply a defective perceptive mechanism? Should all cases of bilateral congenital atresia be operated upon with a view of establishing or improving the auditory function?

Fig. 307.—Microtia.

Fig. 308.—Microtia.

Theoretically, since the labyrinth is developed from the cartilaginous skeleton, and the structures of the conducting mechanism are of distinctly later developement from its enveloping membranes, it is difficult to understand why any congenital defect may not exist in either mechanism independently of the other. But whatever one's personal theory, if one examines the literature bearing on these abnormalities, one finds such diversity of opinion, that one is forced to conclude that many aspects of the subject are still *sub judice*.

The comparatively small number of published reports of cases operated upon by American surgeons leads one to suspect that the results as a rule have not been particularly gratifying. Dr. John R. Page's case[1] of congenital bilateral microtia with atresia of the canals is interesting and

[1] Page, J. R.: Congenital bilateral microtia, etc. Transactions Amer. Oto. Soc. 1914.

instructive in that the anatomical defects revealed by the operation and the surgical method pursued are clearly stated. While the results of hearing tests before and after operation are not given in detail, it is clear that the hearing was greatly improved. But analysis of the operative findings in this case, with the normal caloric reactions and prompt improvement of hearing, shows that the deafness was due to defective sound transmission rather than to a defective labyrinth. Too great optimism as to the average results of surgical intervention would not, therefore, be justified as a logical deduction from this case.

In this country, probably the most comprehensive monograph on this subject is that published in 1917 by Dr. Lee Wallace Dean[2] of Iowa City. This paper includes interesting and extensive references to the literature of the subject with in some cases brief synopses of case reports and the deductions based thereon. Dr. Dean ends his paper with an instructive and unusually complete history of a case from his own practice. This case has so clear a bearing on one mooted point in the interpretation of these congenital defects, that the salient facts will be briefly stated.

The history is that of a boy of 15 years, with auricles of normal size and shape but with complete atresca of both canals. Hearing "poor since birth" and rapidly failing. Hearing defects typical of a disturbance of sound conduction. Operation demonstrated in each ear a movable incus and stapes; no malleus discovered; no annulus or drum membrane. The normal space of the bony meatus was occupied on each side by solid bone. In the effort to establish a practical sound-conducting apparatus this bone was drilled out by an electric burr. To provide an orifice for the artificial canal thus formed a large opening was made in the concha. The canal and small aditus to which it led was later lined with Thiersch grafts, resulting ultimately in complete dermatization. As a result of the operation, which was practically the same on the two sides, the hearing was immensely improved. Hearing distance for conversational voice increased from 3 feet and 5 feet (right and left) to 48 feet. The case is of great importance in demonstrating that atresia of the canals may coexist with perfectly normal labyrinths, and as suggesting that the prognostic value of operative intervention is probably in direct ratio to the amount of hearing before operation.

Apparently one is justified at least in the following conclusions: (1) Bilateral microtia with congenital atresia of the canals and accompanied by extreme deafness—*i.e.*, deafness so pronounced as to render the acquisition of speech doubtful—offers so little definite promise of improvement by any means at our disposal as to make operative intervention of questionable prognostic value. Naturally, in the first 2 or 3 years of life, hearing tests are not very reliable, and one may have to assume risks in operating, as the

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[2] Dean. L. W.: Report of a case of bilateral, congenital osseous atresia of the auditory canals: Transactions American Laryngological, Rhinological and Otological Society. 1917.

only hopeful possibility. Clearly only one ear should be operated at a time. (2) Bilateral microtia or atresia of the canals (or both) with hearing in either ear of sufficient acuteness to suggest an intact or at least functionally useful perceptive mechanism, should be operated upon.

In operative cases, the rational procedure is to open the antrum from behind as for a radical operation, and lower the posterior canal wall, if a canal exists. In the absence of a bony meatus the tympanum should be exposed by very careful removal of the outer wall of the aditus. In the absence of a drum membrane the malleus and incus, if present, should be removed. A bony meatus may then be made, if conditions permit, by removal of bone from within outward. The plastic work for providing a suitable orifice and canal would of course have to depend upon the anatom-

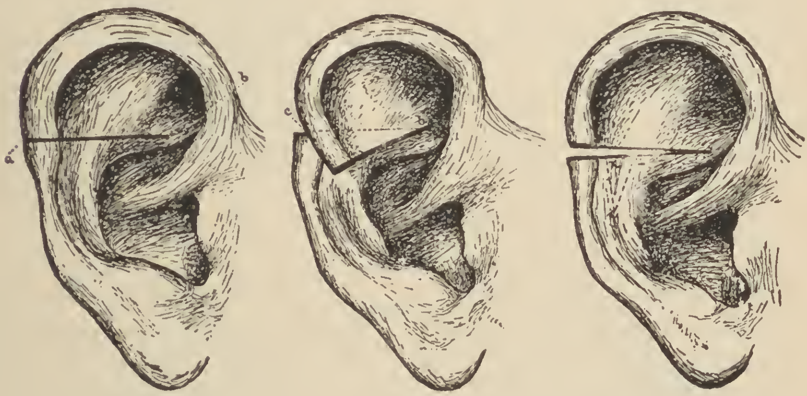


FIG. 309. FIG. 310. FIG. 311.

FIGS. 309, 310, 311.—Usual method of reducing an abnormally large auricle (Goldstein).

ical condition of each individual case. The electric burr may possibly play a useful rôle in these cases, and skin grafting will in all cases be an essential means of maintaining the artificial orifice and meatus.

The treatment of the lesser aural abnormalities is almost wholly surgical. When it is observed that the ears of an infant or very young child, while of normal size and shape, tend to stand out prominently from the side of the head, something may be done to correct or avert the deformity by bandaging the ears to the side of the head at night, the use of nightcaps, etc. Such appliances, however, are uncomfortable, not conducive to sound sleep, and the results are extremely uncertain. We shall consider, therefore, only the surgical treatment of patients who shall have reached an age at which palliative measures are obviously useless.

When the abnormality is chiefly that of unusual size,—*i.e.*, depends mainly upon undue peripheral growth or expansion of the cartilaginous

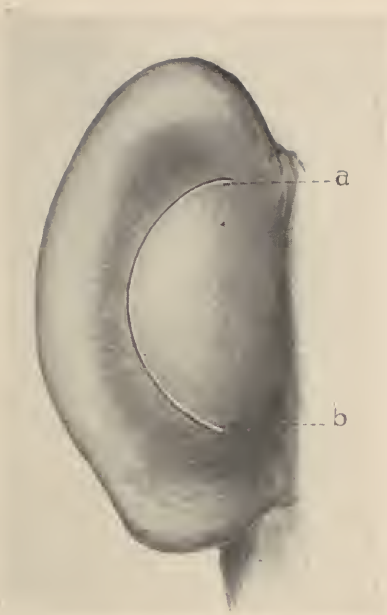
frame,—the deformity may usually be made less conspicuous by an operation fixating the auricle more closely to the side of the head. This in competent hands is a safe procedure,—*i.e.*, not likely to result in perichondritis. Actual reduction of size, on the other hand, can be obtained only as the result of an operation including some removal of cartilage. An operation described by Dr. Goldstein,[3] designed to reduce the vertical diameter of an abnormally long auricle, is clearly shown in the accompanying figures (309, 310, 311).

After the horizontal incision *a–b* (Fig. 309) is made, the upper segment is overlapped upon the lower and drawn down until the desired reduction in size is attained. The redundant wedge-shaped segment of cartilage is then removed by a careful incision through the line *b–c*, Fig. 310. If the peripheral edges do not meet,—*i.e.*, do not form a continuous peripheral margin,—they may be brought into proper line by removal of a second small wedge of tissue from whichever segment extends beyond the other,—*e.g.*, the portion included in the triangle *n–f–z*, Fig. 311. This, however, should usually be avoided. The parts are then united by a line of interrupted silk sutures passing through the cartilage and both skin surfaces and tied on either the posterior or anterior surface of the auricle.

For displacement due to abnormal width of the auricular cartilage, Goldstein has devised an ingenious operation (Figs. 312, 313, 314, 315), for which we append his own description: "The curvilinear incision *a–b* (Fig. 312) is made on the posterior surface of the auricle, almost corresponding with the natural ridge of the antihelix anteriorly. The convexity of this curved incision line presents outward toward the rim of the helix. The flap is shaped so that, when it is dissected upward and over toward the mastoid area, the largest possible surface of cartilage is exposed. The dissection of the integument flap and perichondrium from the cartilage is simple, and, as the adhesion of the perichondrium is not firm, it can be quickly done.

"When the cartilage is cleanly exposed, an incision in the line *e–f* (Fig. 313), curving inward, almost at a right angle at points *e* and *f*, is made through the cartilage, care being taken not to wound the underlying integument which forms the anterior covering of the auricle. With a narrow, blunt elevator, such as is used in the resection of the nasal septum, the two cartilaginous flaps at *c–d* and *e–f* are now carefully elevated from the underlying perichondrium to a sufficient breadth to admit of their manipulation, and to permit the flap at *c–d* to be superimposed upon the flap at *e–f*, when brought together by a series of two mattress sutures, each suture being passed twice through each of the cartilages. When gentle traction is applied in tightening the sutures, the two cartilaginous flaps will be drawn over each other and the width of the auricle reduced in the same proportion as the sutures are introduced from the edges of the respective cartilaginous flaps. The sutures are applied by first passing

[3] Goldstein, M. A.: The Laryngoscope, vol. xviii, No. 10, pp. 835–7.



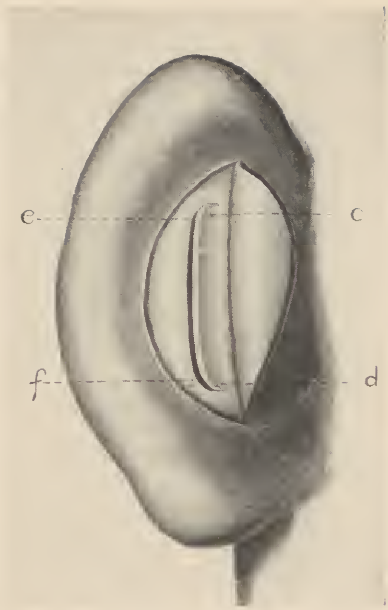
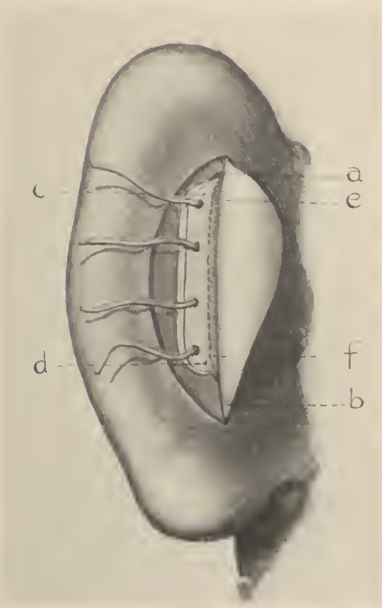


FIG. 312.

FIG. 313.



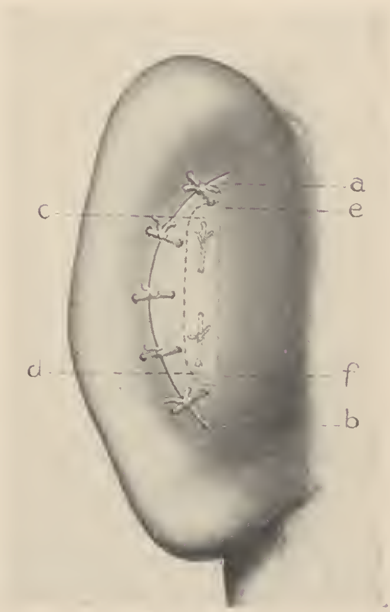


FIG. 314.

FIG. 315.

FIGS. 312, 313, 314, 315.—Operation for reducing abnormally wide auricle. (After Goldstein.)

a sharply-curved needle, armed with thin chromatized catgut, through a point at the upper end of flap *c–d*, then downward through flap *e–f*, then over and under through an opposite point in flap *c–d*.

"Another suture is similarly placed in the lower part of these flaps. The cartilage is tough and will stand considerable traction when the sutures are brought together. The sutures are then tied over flap *c–d* (Fig. 314), the redundancy at the edge of the integument flap *a–b* is then carefully cut away and the integument stitched in position in the original line of incision (Fig. 315). With the traction produced by the overlapping of the cartilaginous flaps, some slight wrinkling or puckering may at first appear on the anterior surface of the auricle, but if the anterior perichondrium and integument have been liberally elevated from the cartilage, this wrinkling of the integument will disappear with the healing of the wound."

In any operation involving incision or removal of auricular cartilage, the chances of failure are considerable. In the first place, one difficulty is the cosmetic necessity of obtaining the same mechanical result in each ear. Healing by first intention is essential to a perfect result. Should infection occur, perichondritis may result. This in turn may lead to inflammation and finally necrosis of the cartilage itself, with resulting deformity far greater than that which the operation was designed to correct.

There are, of course, other operations than the two above mentioned for reducing the size of a large auricle, but, as a rule, they are more complicated, call for multiple incisions of the cartilage, and the chances of infection and perichondritis are proportionately greater.

Removal of Supernumerary Nodules.—The correction of the disfigurement caused by the presence of supernumerary parts or appendages (Figs. 304, 305) consists in their surgical removal. The overlying skin is carefully dissected up, and the cartilaginous nodules or projections forming the basis of the deformity are excised. The skin flaps are then brought together, redundant portions removed, and the wound closed with silkworm-gut sutures.

Treatment of Prominent or Projecting Ears.—In those cases in which the auricle is neither malformed nor abnormally large, and in which displacement is mainly forward, the condition may be corrected by the comparatively simple operation indicated by Figs. 316, 317, and 318.

With tooth thumb forceps the skin is caught up at a point upon the posterior surface of the concha a variable distance from the postauricular fold, and by this the auricle is drawn backward and inward against the corresponding mastoid surface. If this brings the auricle into an approximately normal position, the point grasped by the forceps and that upon the mastoid surface with which it comes in contact are nicked (Fig. 316, *x*, *x*). These coming together represent the common point for fixating the auricle to the side of the skull. A curved line is now marked on the posterior surface of the concha, joining the two ends of which a similar line is marked upon the skin covering the mastoid (Fig. 317). In each case these lines

traverse the contact points (x). We have thus marked out upon the postauricular surfaces of the auricle and mastoid an area from which the skin and soft parts are to be dissected up and removed. Upon the auricular surface the tissues are dissected from the perichondrium and upon the mastoid from the periosteum. In other words, neither the cartilage nor the bone is deprived of its nutritive and protective membrane. When the auricle is now pushed back into normal position, we have a raw surface upon the back of the auricle in contact with a raw surface of similar shape upon the mastoid. The parts are held in apposition by sutures uniting the skin

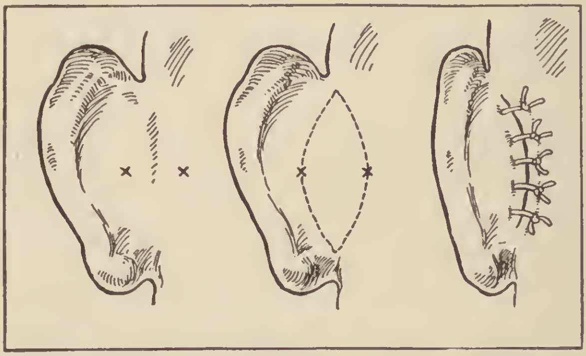


FIG. 316. FIG. 317. FIG. 318.

FIGS. 316, 317, 318.—Operation for the correction of moderate projection.

edges forming the boundaries of the two denuded areas (Fig. 318). Both ears should be operated upon at the same time. Following the operation, a large pad of sterile gauze is placed over each ear, and a bandage applied. At the end of five or six days when the stitches are removed, the auricles are fixated in their new and more correct position by the skin suture line and by the firm adhesion of the broad surfaces from which the skin was removed.

Ruttin's Operation.—Ruttin's operation, involving practically the same principles as those applied in the preceding, is appropriate to cases of more exaggerated displacement, and is proportionately more radical. The essential features are graphically shown in the accompanying illustrations (Figs. 319 to 323).

The auricle is forcibly placed against the side of the head (Fig. 319), and a line is marked upon the postauricular surface corresponding to the centre of the posterior limit of auricular contact (Fig. 319). This line is then made curvilinear and parallel with the postauricular fold, or line of postauricular attachment (Fig. 320). A similar curvilinear incision is next made upon the posterior surface of the auricle just behind the line of auricular attachment, and a curved flap dissected up from the perichondrium (Fig. 321). The upper and lower extremities of the two incisions are now united by horizontal incisions, and the included soft parts dis-

sected up and removed (Fig. 322). The curved auricular flap is finally drawn backward, carrying the auricle with it, and sutured to the corresponding margin of skin behind the ear (Fig. 323). The ear is held against the side of the head by a pressure pad and bandage. When healing is complete and the stitches are removed, the auricle is held in the new and less conspicuous position by the suture line and by the adhesion of the broad surfaces denuded in the operation.

FIG. 319.

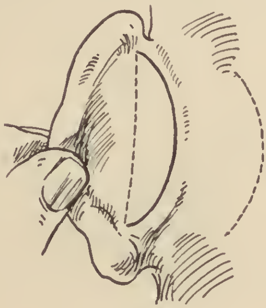


FIG. 320.

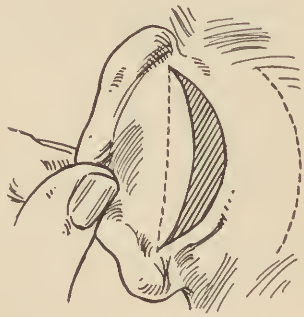


FIG. 321.

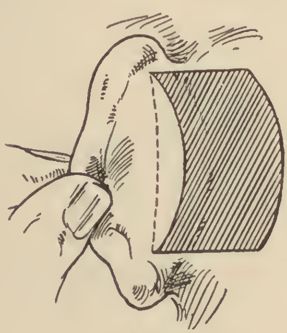


FIG. 322.

FIG. 323.

FIGS. 319, 320, 321, 322, 323.—Ruttin's operation for projecting auricles.

This operation is practical, and its results should be permanent. A possible disadvantage is the obliteration of the space between auricle and scalp behind the ear.

Duel's Operation.—Duel's operation is an effort to correct a forward and outward displacement of the auricle without obliteration of the postauricular fold or sulcus.

By means of two thumb forceps the skin covering the posterior surface of the auricle is caught up at two points (Fig. 324, *x*, *x*), by which the auricle is drawn inward against the side of the head. The skin points caught by the forceps are brought into contact with the side of the head along a more or less vertical line a little behind the postauricular fold.

Different points upon the posterior surface of the pinna are tried until two are found which, when brought into appropriate contact with the side of the head near the attachment of the auricle, maintain the auricle in proper position. These contact points are then marked,—those on the posterior surface of the pinna being pinched with the forceps, while those behind the ear are nicked. The four contact points thus marked are united by three incisions, two horizontal and one vertical (Fig. 324), thus outlining a quadrilateral flap which must be dissected up as indicated in Fig. 325. About a half inch behind and parallel with the first vertical incision, a

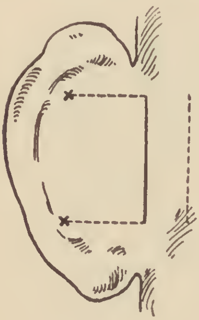


Fig. 324.

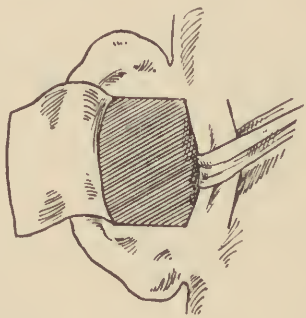


Fig. 325.

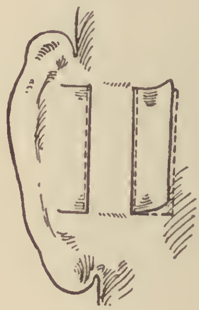


Fig. 326.

Fig. 327.

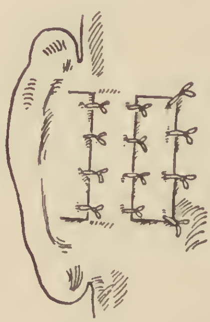


Fig. 328.

Figs. 324, 325, 326, 327, 328.—Duel's operation for projecting ears.

second incision is made outlining between them a narrow strip, which, when dissected from the underlying bone, forms a movable band attached only above and below (Fig. 325). Beneath this band the quadrilateral flap is now introduced, and, when rather forcibly drawn back, as shown in Fig. 326, increases the auricular bend at the antihelix and draws the auricle into position against the side of the head. When the deformity is caused by the absence of the usual bend of the antihelix, this strapping of the ear inward is designed to create such a bend by folding the auricular plate upon itself. Having drawn the quadrilateral flap beneath the ver-

tical band and well backward, we have the middle third of the flap hidden beneath the vertical band, and the posterior third extending well behind it as in Fig. 326. The next step is to mark out upon the postauricular surface the narrow quadrilateral area lying beneath the posterior end of the flap, throughout which the skin and soft parts must be removed. The skin must also be removed from the middle third of the flap,—*i.e.*, that part of it which is destined in the completed operation to lie under the vertical band. These steps completed are shown in Fig. 327. The flap is now again drawn under the vertical band until the denuded area is in contact with the under surface of the latter, and is then secured in position by the three vertical rows of interrupted sutures shown in Fig. 328. For this operation sutures of fine silk are the best. During all these manipulations the vertical band must be held in position by an assistant; and after the final sutures are placed, a compression pad is necessary to keep the various raw surfaces in appropriate apposition and contact.

Duel's operation is more complicated, more difficult, and in the hands of the average surgeon its results are therefore more uncertain than those of the two preceding operations.

CHAPTER XXI.

NON-SUPPURATIVE DISEASES OF THE LABYRINTH.

Ménière's Disease.—Definition: This term may be properly applied only to the condition in which pronounced deafness and the phenomena of vestibular irritation occur suddenly as a result of hemorrhage into the labyrinth.

In its true form it is unquestionably one of the rarest of lesions. Ménière has given a clear description of the first case observed clinically and coming to autopsy.[1] A young girl, during a menstrual period, was attacked with sudden extreme vertigo, nausea and vomiting, loud tinnitus, and absolute deafness. Death occurred on the fifth day of the attack. Post-mortem examination showed the presence in the semicircular canals of a thick bloody exudate, only a trace of which could be found in the vestibule, and which was not present in the cochlea. The most careful examination of the brain and cord failed to reveal any abnormal condition, and the exact cause of death was not determined.

Etiology.—While many conditions—including pronounced anæmia, leukæmia, rheumatism, nephritis, intense heat, and prolonged exposure to the sun—are mentioned as among the possible causes, cases of the disease, typical clinically and verified by autopsy, have not been observed in sufficient number to justify any conclusive statements. Politzer mentions that cases presenting all the clinical features of the disease have died in which the post-mortem findings have failed to provide any evidence of hemorrhage into the canals, and, *per contra*, a hemorrhagic exudate within the vestibule and canals has more than once been observed post mortem in cases in which no history of typical Ménière's symptoms during life could be obtained. Furthermore, Ménière's case, upon which our conception of the disease still very largely rests, does not supply a logically complete picture; for one of the conspicuous clinical features of the attack was the sudden and profound deafness, yet his post-mortem examination revealed no abnormality within the cochlea.

In considering this lesion as a pathologic entity, it must be remembered that many conditions which we now know to be capable of inducing identically the same clinical phenomena were practically unrecognized until a comparatively recent date. It is probable, therefore, that many cases have in the past been recorded as true examples of Ménière's disease which have in reality been the result of other conditions, the nature of which would now be easily recognized. The disease is still more or less wrapped in mystery, which future investigations may serve to clear up.

Diagnosis.—According to our present concept of the disease, the

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[1] Ménière: Gazette médicale de Paris, 1861, p. 598.

diagnosis depends not only upon the clinical features of the attack, but demands also a knowledge of the condition of the patient's ears before and immediately following the attack. To justify a reasonably positive diagnosis, the attack must occur in an individual whose ears are known previously to have been normal or approximately so, and inspection of the ears shortly after the onset must show absence of any pathologic changes within the tympanum upon which an hypothesis of possible labyrinthine infection might be based.

The SYMPTOMS are those characteristic of sudden, intense irritation of the vestibular apparatus plus ablation of the cochlear function. The attack begins with severe subjective vertigo, great disturbance of static equilibrium, loud tinnitus, nausea and vomiting, and pronounced deafness in one or both ears. The patient does not, as a rule, lose consciousness. At the onset he is forced by the urgency of the symptoms to go to bed. Sudden movements of the head or the attempt to rise aggravate the subjective symptoms and may induce vomiting. Nystagmus without doubt is an invariable phenomenon of the onset.

As with every disorder depending upon an organic lesion, the severity of the symptoms varies with the extent and character of the lesion. It is conceivable that very slight hemorrhage, confined to one or more of the canals, might induce very pronounced and distressing symptoms, which, however, might disappear with the final absorption of the exudate. Only in this way can be explained certain very typical recorded cases in which recovery has been fairly rapid and apparently complete.

A form of the disease described in most text-books under the term "apoplectic form of Ménière's disease" is characterized by a similar train of clinical phenomena plus symptoms of cerebral disturbance, the chief of which being loss of consciousness. The loss of consciousness follows closely upon the onset and is of short duration, leaving the patient at first in a condition bordering upon collapse, from which he gradually recovers. The exact pathologic basis of such seizures is as yet altogether hypothetical. It is possible that they may eventually be proved to be quite unrelated pathologically to true Ménière's disease.

The PROGNOSIS depends on the extent of the lesion. It is conceivable that a very slight extravasation of blood might be absorbed, leaving little or no disturbance of function. On the other hand, the membranous labyrinth is known to be a structure so extremely sensitive to any form of direct mechanical injury that it is difficult to believe that a considerable bloody exudate could fill any of the bony compartments of the labyrinth without causing practical destruction, or at least permanent ablation of function, of the membranous structures contained therein. It is one of the inconsistencies of our conception of the disease that, while the semicircular canals are looked upon as the characteristic site of the lesion, permanent deafness is said in severe cases to be one of the most constant and characteristic sequelæ.

TREATMENT.—The logical treatment is that of any irritative lesion of

the labyrinth plus measures appropriate to any form of concealed hemorrhage. Absolute rest in bed should be enforced during the first two or three days of the attack. It might be well, unless uncomfortable to the patient, to have the head slightly raised. A laxative should be administered and particular attention directed to keeping the bowels freely moved. During the first twenty-four or forty-eight hours fluid diet is indicated. Applications of dry heat to the feet, calves of the legs, and abdomen, and cold to the head have been advised, probably on the hypothesis of a pathologic analogy between the lesion and cerebral hemorrhage. They probably are without influence either upon the lesions or the resultant symptoms.

For the various drugs advised I can see no indication, unless it be the iodide of potassium, small doses of which, after the acute stage of the attack has passed, may tend to promote absorption of the fibrinous exudate which is the pathologic basis of the disease.

"Vertigo ab aure læso."—A term which time-honored custom has transmitted from one text-book to another, but which in the author's opinion should long since have been dropped from otological literature and parlance, is "vertigo ab aure læso." It is applied to the short attacks of intense vertigo, ataxia, tinnitus aurium, and vomiting which occur as a rare accompaniment of cases of otosclerosis or chronic hyperplastic otitis media, and which have been supposed to be in some way related to mechanically induced changes in intra-labyrinthine pressure. A careful analysis of the symptoms described shows that they coincide exactly with the usual phenomena of vestibular irritation, the one characteristic feature not invariably mentioned being nystagmus. In the light of recent advances in our knowledge, a close study of the symptoms will show these attacks to depend upon causes not easily traceable to a tympanic lesion, or at least to accord better with other theories of origin. The writer does not wish to belittle the value of the clinical observations recorded under this name, but simply to protest against the retention of a term which our advancing knowledge of labyrinthine phenomena should have rendered obsolete. These cases present perfectly characteristic manifestations either of direct irritation of one or both labyrinths, or of the excitation of one labyrinth as a result of sudden paralysis or ablation of function of the opposite organ. Each case should be subjected to the most careful observation and study, and an attempt made to determine exactly the underlying cause. "Vertigo ab aure læso" is an inexact term which has supplied a convenient cloak for a gap in our knowledge.

Leukæmic Deafness.—The so-called leukæmic deafness is the result of certain morbid changes within the labyrinth which occasionally occur in cases of leukæmia. Politzer, who reported the first authentic case of leukæmic labyrinthitis in 1885, cites Vidal and Isambert as having estimated that the aural lesion occurs in 10 per cent. of all cases of leukæmia.

The most characteristic pathologic change is the presence of a fibrinous, bloody exudate and masses of lymph-cells in both the perilymph and endolymph spaces of the labyrinth. Both the cochlear and vestibular

sections of the labyrinth are usually involved. In Politzer's case masses of "leukæmic plaques consisting of lymph-cells" were found within the scala tympani resting upon the basilar membrane and bony wall of the canal. Somewhat similar post-mortem findings have been reported by other observers. In England the disease has been studied by Richard Lake, who has been able to exhibit a number of characteristic pathological specimens of the lesion.

Symptoms.—Two clinical types of the disease have been recorded: (1) Those in which the symptoms were obviously the result of sudden hemorrhage into the vestibule and canals, and therefore resembled closely a typical attack of Ménière's disease,—*i.e.*, intense vertigo with attendant ataxia, loud tinnitus, and deafness; and (2) cases in which hemorrhage has played a minor part, the characteristic change being an infiltration of the endosteal lining of the bony spaces involved, and the deposition of masses of lymph-cells within both the perilymphatic and endolymphatic spaces of the labyrinth. Great thickening of the endosteum has been noted in some cases. Such a lesion would lead logically to a more gradual and less spectacular development of symptoms,—*e.g.*, impairment of hearing of labyrinthine type, progressing rapidly to an extreme grade of deafness. Symptoms of disturbed vestibular function also characterize this form of the disease, but are much less pronounced than in the hemorrhagic type of the lesion.

Anæmia of the Labyrinth.—It is not always easy to trace with any certainty the symptoms due to anæmia of the labyrinth to their source. It is known, however, that in cases of profuse traumatic hemorrhage, in parturition,—in fact, in any condition causing sudden withdrawal from the body of large amounts of blood,—loud tinnitus aurium, intense dizziness, and in some cases vomiting, are present. In such cases the tinnitus and vertigo, and in all probability the vomiting, seem to be clearly of vestibular origin, and are explainable upon no other hypothesis than that of sudden diminution of the vestibular blood supply.

The attacks of vertigo and tinnitus which occasionally occur during the course of wasting illnesses—*e.g.*, severe typhoid fever—may be due either to direct irritation of the vestibular structures by toxic substances circulating in the blood or to the participation of the labyrinth in the anæmia which is a logical result of the disease. On the other hand, the extreme dizziness, particularly if accompanied by loud tinnitus, which may attend the first attempt of a convalescent from any wasting disease to assume the upright position,—*i.e.*, to stand,—would seem to be the logical expression of a sudden diminution of the vestibular arterial supply. These are instances of labyrinthine disorder which rapidly correct themselves as the patient regains his normal condition. They are cited as convincing examples of aural disorders directly depending upon labyrinthine anæmia.

More difficult of positive diagnosis—impossible of correct interpretation except through a very careful and thorough process of exclusion, but of undoubted occurrence—are cases of vertigo with auditory impairment

resulting from the severer forms of anæmia. The symptoms are similar in kind to those of vestibular irritation through other agencies,—*i.e.*, vertigo, marked disturbance of static equilibrium, subjective noises, and loss or impairment of hearing. One diagnostic sign of the origin of such an attack (Dundas Grant) is said to be the relief of symptoms when the patient assumes the recumbent position. Greater dependence, however, must be placed upon blood counts and the exclusion of other causes.

There are two considerations which make it of particular importance that cases belonging to this class should be correctly diagnosticated,—viz., (1) only a determination of the underlying cause will lead to an effective plan of treatment, and (2) failure to institute the proper treatment may leave the patient in the most favorable condition for the development of otosclerosis (H. A. Gray, see pages 249–250).

Treatment.—The treatment is obviously constitutional rather than local. Rest, abstention so far as circumstances may admit from business cares and worries, and the administration of constructive tonics are our chief aids in relieving such patients.

Syphilis of the Labyrinth.—Four clinical types of labyrinthine syphilis are recognized,—viz., (1) The congenital type, seen in its most characteristic and easily recognized form in children exhibiting other stigmata of the disease. The aural lesion is usually bilateral, though one ear may be, and usually is, more seriously impaired than the other. Examination of the ears may reveal physically normal drum membranes, or the physical signs of middle-ear suppuration may render the syphilitic character of the labyrinthine affection less easy to recognize. The deafness is of distinctly labyrinth type and usually of advanced grade. Examination of the child shows in a certain percentage of cases the characteristic notched condition of the teeth (Hutchinson's). According to Fraser,[2] this defect occurs in 50 per cent. of cases: Interstitial keratitis is another defect occasionally met with, as also are destructive lesions (defects) of the soft and hard palate. With or without these defects, the child not infrequently presents a stunted, prematurely old appearance which is more or less characteristic of these little unfortunates. The above abnormalities mark the most pronounced and therefore conspicuous form of congenital syphilis. There are many cases, however, in which the child does not exhibit such obvious stigmata, in which case a thorough physical examination, a careful analysis of the family history, and resort to the Wassermann test may be necessary to establish a correct diagnosis.

The prognosis, so far as the auditory function is concerned, is hopeless.

(2) The second variety of syphilitic labyrinthitis is that which occasionally complicates the acquired form of the disease in adults. It develops most frequently in the late secondary or in the tertiary stage, rarely in the primary stage of the disease. It is characterized clinically by very sudden and usually very extreme deafness in one or both ears. That the vestib-

[2] Fraser: Journal of Laryng., Aug., 1909.

ular apparatus is also involved in a very large percentage of cases is shown by the frequent evidences of disturbed equilibrium,—*e.g.*, nystagmus, vertigo, static ataxia, etc. When the lesion is unilateral and completely annuls the function of the affected vestibular mechanism, the onset is necessarily characterized by all the usual phenomena of vestibular irritation or excitation. When both labyrinths are simultaneously paralyzed, the patient exhibits the type of vertigo and ataxia characteristic of vestibular paralysis. The writer has recorded the history of such a case,[3] in which the patient was absolutely deaf in both ears, and both vestibular mechanisms were absolutely non-irritable by either rotation or the caloric test.

The clinical features of these cases usually leave in doubt the question as to whether the membranous labyrinth or the eighth nerve is the structure primarily attacked. Involvement of other cranial nerves and simultaneous and equal disturbance of both cochlear and vestibular functions would point to the nerve as the structure primarily attacked; while the escape of other cranial nerves plus unequal disturbance of the two branches of the eighth nerve would point rather strongly to a direct invasion of the labyrinth.

Aside from the very characteristic onset of the attack in a person whose drum membranes may show no morbid changes, there are usually other evidences of the disease in cuticular eruptions, mucous patches, etc., which, in conjunction with the history of an initial lesion, may leave no doubt as to the syphilitic origin of the labyrinthine disorder.

Though necessarily causing great anxiety, the prognosis is more favorable than in any other form of syphilitic labyrinthitis. The treatment, which is practically the same as for syphilitic affections of the cranial nerves, will be discussed somewhat fully in the chapter devoted to the use of salvarsan in aural disease (see Chapter xxi).

(3) Another clinical variety of the disease, the pathologic basis of which is not always so easily determined, is found in adults who have suffered from chronic constitutional syphilis over a period of years. The drum membranes may or may not show evidences of tympanic disease, which, if present, may represent an intercurrent and pathologically independent lesion. Such a patient frequently gives a history of occasional attacks of vertigo of more or less pronounced type, or such attacks, having belonged to an earlier stage of the disease, may have ceased to annoy him. The diagnosis depends chiefly upon the labyrinthine character of the deafness,—*i.e.*, diminished bone conduction, negative Rinné, loss of hearing for the highest musical tone,—plus constitutional evidences of the disease. Very frequently the pharynx and nasal cavities show the cicatrices and perforations representing the typical lesions of an earlier stage.

Naturally the history of such a patient might be expected to furnish data of considerable diagnostic significance. To other manifestations of the disease the Wassermann test contributes the strongest corroborative evidence.

The lesion from which it is in some cases most difficult to differentiate

[3] Kerrison: Vertigo of Vestibular Paralysis, Transactions Amer. Otol. Soc., 1911.

chronic syphilitic labyrinthitis is that form of otosclerosis which involves not only the labyrinthine capsule but also the membranous cochlea. Between these two conditions a positive differentiation is not in all cases possible. The history of a very sudden development of the deafness, coming on in the late secondary or in the tertiary stage of acquired syphilis, would go far toward establishing the syphilitic character of the labyrinthine lesion.

The treatment in these cases is the treatment of chronic syphilis. The prognosis, so far as any marked improvement of hearing is concerned, is unfavorable.

(4) The fourth clinical type may be dealt with briefly. It is found in patients who are victims of constitutional syphilis of long standing, and whose ears exhibit the physical characteristics of a middle-ear suppuration (usually of offensive type) and the functional reactions of labyrinthine disease. The syphilitic nature of the labyrinthine lesion is usually an hypothesis not susceptible of definite proof. It is, however, an hypothesis of very practical importance, for the reason that improvement, either functional or of the concomitant suppurative lesion of the middle ear, is not likely to result from any plan of local treatment not reinforced by active measures to eradicate the constitutional disease.

Treatment.—There is no manifestation of syphilis which calls more urgently for vigorous efforts to eradicate the systemic poison than syphilitic labyrinthitis. The question of treatment resolves itself into a comparison of the few drugs of known value, and the best methods of administering them. The more important phases of this subject are discussed in the chapter devoted to the use of salvarsan in aural therapy.

Tuberculosis of the Ear.—Tuberculous lesions of the ear usually attack first the tympanum, and such lesions, more than any other form of middle-ear disease, are prone to cause extensive caries or necrosis of the labyrinthine capsule. It would be quite illogical, therefore, to speak of tuberculosis of the tympanum and of the labyrinth as distinct pathological entities, or to consider them under separate headings.

The tubercle bacilli may reach the ear by the blood or lymph channels, but unquestionably the commoner pathway is by the Eustachian canal.

The predisposing causes are (1) inherited predisposition to tuberculosis; (2) environment, *i.e.*, unfavorable surroundings and conditions of life; and (3) age, children being more subject than adults to the disease.

The influence of heredity is shown in the case of individuals inheriting the so-called tubercular diathesis, who undoubtedly are more likely to develop primary tuberculous lesions of the middle ear, and in whom the common type of suppurative otitis media is more likely to be converted into a tuberculous lesion than is the case with an individual of normal resistance.

The influence of environment is seen in the comparative frequency of tuberculous middle-ear lesions among the poorer class of tenement-house dwellers, who have to contend not only with overcrowding and poor ventilation, but often also with the effects of poor and insufficient food. The influence of such conditions is twofold,—*i.e.*, (a) by reducing the

individual's power of resistance to any form of disease, and (b) by his unavoidable proximity to, and association with, tuberculous individuals not suspected of harboring the disease.

Age is a predisposing factor to the extent that infants and young children are particularly subject to tuberculous affections of the middle ear. The greater predisposition of the child as compared with the adult is illustrated again in the children of the tenements, and particularly when they are attacked by one or other of the acute infectious diseases,—*e.g.*, scarlet fever, measles, etc. The very rapid and extensive destruction of the drum membrane and of the bony structures of the tympanum so frequently seen in children suffering from scarlet fever is undoubtedly explained in many cases by a tuberculous element in the pathology of the tympanic lesion. To some extent the greater vulnerability of the very young child to tuberculous middle-ear disease is due to certain anatomical peculiarities of the infantile type of Eustachian tube (see page 154), in consequence of which the middle ear of the child is more open than in the adult to the entrance of infective material from the nasopharynx.

If we will picture to ourselves a condition by no means uncommon in crowded cities,—*i.e.*, of a child, ill-nourished and reduced by unhealthy surroundings; breathing an atmosphere in which tubercle bacilli are probably often present; the presence of adenoids in which many germs, and perhaps tubercle bacilli, find lodgment; add to this a possible inheritance of predisposition to tuberculous infection, and an igniting spark in the form of an acute infectious disease, and we have an ideal condition for the development of a primary tuberculous lesion of the tympanum.

Among adults tuberculosis of the middle ear occurs most frequently in patients in advanced stages of phthisis. It occurs, however, even during the earliest stages of pulmonary tuberculosis, and authenticated cases have been recorded in which no other tuberculous lesion could be determined.

Pathology.—A tuberculous attack upon a previously healthy ear is marked by the following changes: The tympanic mucosa is swollen or œdematous, this condition involving the lining membrane of the promontory and contiguous bony surfaces and to some extent the inner lining of the drum membrane. Small circumscribed thickenings, seen here and there, represent the tubercle nodules. Similar nodules are formed upon the drum membrane, and partly explain one of the characteristic clinical features of the disease which will be referred to later. Under the microscope, sections of the diseased mucosa show widespread infiltration with small cells, with here and there groups of giant cells, and a varying number of tubercle bacilli. This represents a fairly early stage, which is followed, usually rather quickly, by breaking down of the nodules and the formation of granulations which under the microscope are seen to contain giant cells and usually tubercle bacilli. Breaking down of the nodules upon the drum membrane is followed by circumscribed points of ulceration which may involve the entire thickness of the membrana tensa, thus giving rise to the multiple perforations which form so characteristic a feature of the disease.

Following the breaking down of the nodules upon the bony wall of the tympanum, the mucous membrane undergoes rather rapid and widespread destruction, and caries of the underlying bone is a frequent and somewhat characteristic phenomenon. It is also highly characteristic of middle-ear tuberculosis that osseous necrosis spreads rapidly and widely, while the usual clinical phenomena of pain, bone tenderness, and fever are either absent or infinitely less pronounced than usually attend the rapid extension through bone of a suppurative process of streptococcic origin. Another distinctly characteristic feature of tuberculous tympano-mastoiditis is the tendency to the formation of large sequestra, which, if operative intervention is withheld, detach themselves and occasionally are extruded through the auditory meatus or through a postauricular sinus. Still another pathologic feature, which is characteristic of this lesion, is the frequency with which the disease involves large portions of the labyrinthine capsule without producing any of the usual phenomena of vestibular irritation. The Fallopian canal is logically a vulnerable structure in the presence of such a lesion, and facial paralysis, dependent upon tuberculous changes actually involving the nerve itself, is a common feature of advanced middle-ear tuberculosis. While the formation of granulations within the tympanum is characteristic of an early stage of the lesion, they may disappear later, leaving bare tympanic walls presenting a bloodless appearance.

The above account describes in brief the characteristic changes in a case of aural tuberculosis in which infection has occurred by way of the Eustachian canal. There is, however, another mode of invasion, in which the mastoid is apparently the primary point of attack, the tympanic structures being involved later. Bryant explains such cases as being due to a tuberculous infection of the lymphatic glands in the region of the mastoid. Described in stages, the pathologic changes following this mode of infection are said to occur in the following sequence: (a) Enlargement of lymphatic glands over mastoid; (b) postauricular periostitis and œdema; (c) cortical bone involvement with moderate bone tenderness, and (d) rapid extension of the lesion through the intercellular structures of the mastoid to the middle ear.

Symptoms.—The patient, usually without prodromal symptoms, may experience moderate pain or only a sense of discomfort in the ear, or the early changes may occur without any symptoms sufficiently pronounced to attract his attention. In the latter case his first intimation of aural disease may be a slight discharge from the ear. The discharge at first is usually watery, turbid, or slightly bloody. If the ear is examined before rupture has taken place, it will usually present an œdematous appearance, sometimes showing moderate congestion, but rarely the angry red color characteristic of the ordinary forms of tympanic inflammation. Here and there over the membrana tensa are seen small millet-seed projections, pearl-gray in color, representing tuberculous nodules. At a later stage one or more of these nodular points will be seen to have given place

to circumscribed perforations, which still later are apt to become confluent, resulting ultimately in widespread or almost complete destruction of the drum-head. When the lesion is well advanced, bare bone may usually either be seen upon the inner tympanic wall or detected beneath flabby granulations by means of the probe. During all of the above changes the patient may experience practically no aural pain. As the disease advances, involvement of the labyrinthine capsule may be shown by impairment of hearing, and of course in some cases by disturbances of equilibrium. Facial paralysis is said to occur in about 50 per cent. of cases. The lymphatic glands in the region of the mastoid and in front of the tragus are enlarged in a majority of cases. Later the glands in front of the sternomastoid muscle are also involved. In the later stages of the disease, the aural discharge is usually more profuse and also more offensive. The characteristic disintegration of tissue may extend to the ossicular ligaments, as a result of which the ossicles may fall out. Crockett has reported cases in which the malleus or incus has come away with the solution used in irrigating the ear; and Seymour Oppenheimer has recorded a case in which the patient, attempting to wipe out the ear with a cotton-wound applicator, found adhering to the cotton the entire ossicular chain.[4]

Diagnosis.—To epitomize: The diagnosis depends on the comparatively painless onset; the early œdematous, rather than acutely inflamed, appearance of the drum membrane; the appearance upon the drum-head of minute, grayish nodules, giving place later to multiple perforations; the rapid destruction of drum membrane and of the tympanic mucosa; the frequent presence of facial paresis or paralysis; the presence of glandular swelling over the mastoid, and particularly of a small glandular swelling in front of the tragus (Crockett). During operations upon the mastoid, the evidences of very extensive osseous necrosis, with the presence of large and partially detached sequestra, are highly characteristic either of a lesion tuberculous from its incipiency, or of a mixed infection in which a tuberculous element is the dominating factor.

Naturally a decisive diagnostic point in all cases of tuberculosis of the middle ear would be the presence of tubercle bacilli in the aural discharge. It is often difficult or impossible, however, to demonstrate the bacilli in the thin, watery secretion of the early stages of the disease, so that absence of demonstrable bacilli can not be taken as disproof of the disease.

Terminations.—It is difficult to speak authoritatively of the terminations of tuberculous disease of the tympanum, for the reason that tuberculosis primary in and confined to the middle ear and mastoid usually responds favorably to early treatment, whereas a tympanic lesion secondary to pulmonary tuberculosis usually succumbs to the pulmonary lesion. It is an interesting and somewhat unaccountable fact that tympanic tuberculosis rarely leads to tuberculous meningitis, though such extension is known to occur (McCaw).

[4] Oppenheimer, S.: Pathology, Diagnosis, and Treatment of Tuberculosis of the Middle Ear, Medical Record, Nov. 24, 1900.

A question which in the past has been the subject of considerable controversy is whether tuberculosis of the middle ear can occur as a primary lesion or is always secondary to a lesion in the lungs or elsewhere in the body. Apparently there is no longer reason to doubt that primary tympanic lesions do occur, and that under skilful treatment they offer a fairly good prognosis. In Europe the work of Habermann and of Milligan has thrown considerable light on this question. In America, cases of tuberculosis confined to the ear have been reported by Goldstein, Crockett, McCaw, and others, and a fair proportion of these cases have recovered wholly, and apparently permanently, under prompt and wise surgical treatment. While children furnish a majority of the cases of primary middle-ear tuberculosis, adults are by no means immune. Two of Goldstein's four cases occurred in adults.[5] McCaw's[6] case was that of a man who, without at any time presenting symptoms of pulmonary tuberculosis, contracted a tuberculous lesion of the left middle ear. Clinically the disease presented all the classical features of tympanic tuberculosis, ending, however, in tuberculous meningitis, a somewhat rare termination. Operation showed widespread osseous necrosis and the dura in the region of the destroyed tegmen antri greatly thickened and studded with miliary tubercles. Autopsy showed lungs, liver, spleen, and kidneys free from any signs of tuberculous change and the mesenteric glands not enlarged. This apparently was a primary tuberculous lesion of the ear, to which the disease was confined up to the fatal implication of the meninges. Bryant[7] has recorded an exceedingly interesting case in which a tuberculous lesion of the middle ear was positively diagnosticated in a man, apparently in robust health, who, though frequently examined, presented no symptoms of pulmonary tuberculosis until six months later. Bryant's interpretation of this case is that the pulmonary lesion was the primary focus, from which hypothesis he makes the deduction that one may be able to diagnosticate pulmonary tuberculosis from tuberculous changes in the ear long before pulmonary symptoms are present. There is nothing in Bryant's history of the case, however, to prove this sequence, and there seems no reason why the hypothesis may not be reversed,—*i.e.*, that the aural disease was the primary focus to which the pulmonary lesion was secondary.

Prognosis. — In a majority of adult cases, the tympanic lesion is secondary to an advanced stage of pulmonary phthisis, in which case the prognosis is wholly unfavorable. When a primary tympanic lesion is diagnosticated early and brought under rational treatment, the chances of recovery are fairly good. Crockett,[8] who has reported a small but

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[5] Goldstein, M. A.: Primary Tuberculosis of the Ear, etc., Medical News, March 14, 1903.

[6] McCaw, J. F.: Tuberculous Otitis Media in an Otherwise Healthy Adult, Medical News, Oct. 12, 1901.

[7] Bryant, W. S.: Tuberculosis of the Ear, Medical Record, Sept. 26, 1908.

[8] Crockett, E. H.: Tuberculosis of the Middle Ear and Mastoid, Jour. Amer. Med. Assoc., Oct. 20, 1906.

exceedingly interesting series of cases in children, records his belief that primary tuberculosis of the middle ear and mastoid should be treated by prompt surgical removal of all diseased bone, under which plan of treatment he regards the prognosis as quite as favorable as in the severe mastoid lesions of suppurative type complicating scarlet fever.

TREATMENT.—Aside from local cleanliness, it is perhaps best not to place undue emphasis upon the local treatment of these cases. It is a recognized fact that syringing the ears is injurious in tuberculous otitis media. The cleansing must, therefore, for the most part be accomplished by wiping the ear out with sterile cotton. Powders—*e.g.*, of boric acid, xeroform, etc.—help to keep the ear clean and dry. The throat and nose should be kept as free as possible from pathogenic bacteria by the use of antiseptic sprays and gargles.

Quite as important as the local treatment is the management of the patient's life. Change of climate, the arrangement of the sleeping apartment, freedom from care and nerve-strain, life in the open air, the use of constructive tonics and the most nutritious and easily digested diet play as important a rôle as in pulmonary tuberculosis; for they not only influence the outcome of the aural lesion, but also go far to prevent secondary invasion of the lungs, a consideration which should never be lost sight of in dealing with primary tuberculous lesions of the ear.

As to the question of operating, that should be decided only after a consideration of all the facts bearing upon each particular case. In the first place, it is well to bear in mind that many cases of middle-ear tuberculosis have recovered wholly under rational constitutional and hygienic management, with no other local treatment than measures for cleansing the ears. Crockett has recorded several instances of apparently complete recovery under such conservative methods of treatment. On the other hand, in the presence of a rapidly-spreading bone lesion primary in and confined to the ear, there would seem to be no question that thorough eradication of all diseased bone by a careful Schwartze-Stacke operation provides the only logical course and offers the best promise of recovery.

Undoubtedly the most difficult problems may be presented in cases in which tympanic and pulmonary lesions coexist. With pulmonary phthisis well advanced, it is clear that a mastoid operation, even though otherwise clearly indicated, may involve great risks in the influence it might exert upon the progress of the lesion in the lung. The correct course to pursue in such cases does not provide a favorable theme for dogmatic statement. If the pulmonary lesion has reached a stage at which recovery may not be hoped for, a mastoid operation would be quite unjustifiable. On the other hand, cases undoubtedly occur in which a timely opening of the mastoid would constitute an essential step in the treatment of pulmonary tuberculosis.

CHAPTER XXII.

OTOLOGIC LESSONS OF THE WORLD WAR.

WAR DEAFNESS: DEAFNESS DUE TO DIRECT LABYRINTHINE INJURY: CONCUSSION DEAFNESS: PSYCHIC DEAFNESS: PITHIATISM SIMULATION: PITHIATIC DEAFNESS: DIAGNOSIS AND RATIONALE OF TREATMENT.

Probably most of us have heard the remark that the war has added little to our knowledge applicable to the peace-time practice of medicine. This, for the moment, may be true, since the men whose opportunities for observation and deduction have been greatest have hardly yet had time to correlate and systematize their experiences. Even less has the profession at large had time to digest and assimilate the published facts.

As to the surgical experiences of the war, only those who have actually observed or practiced it are in a position at the present time to speak. Yet, whoever reads even such fragmentary accounts as have so far reached us will surely come upon statements of actual experience, reflections on phenomena observed, interpretations of unfamiliar syndromes, modifications of classical surgical procedure, etc., etc., which cannot fail to stimulate thought and awaken a desire to review our time-honored theories and methods in the light of the accumulated data of the war. To illustrate my meaning:—let us take for example a comparatively simple accident, *i.e.*, the linear or stellate rupture of the drumhead occasionally resulting from a forcible slap, or "box on the ear." We are accustomed to regard the rupture of the membrane, on account of its possible sequelæ, as the most serious result of this outrage. But in his study of cases of deafness produced by the concussion of loud and near-by explosions, Bourgeois has observed that in those cases in which the drum membrane is ruptured, the injury of the perceptive labyrinth and the consequent deafness are less grave and less apt to be permanent than in cases in which the drum membrane remains intact. The logic of this observation is clear: for if the first force of the air condensation is expended in rupturing the drumhead, it is obvious that this structure cannot carry the hammer handle and with it the incus and foot-plate of the stapes suddenly and forcibly inward, and the insult to the membranous labyrinth is lessened. A further conservative influence is the fact that, with the rupture of the drumhead, air entering the tympanum, acts simultaneously on the stapes and membrane of the round window, thereby destroying their compensatory function and thus lessening or inhibiting the movements of the labyrinthine fluids. We must, therefore, hereafter regard the accident to the drumhead following a blow on the ear as an unfortunate accident but, nevertheless, as the lesser of two possible evils. Again, Bourgeois formulates a rule as fully established by the experiences of the

war that accidents to the ear resulting in rupture of the drum membrane *should not be irrigated nor treated with bactericidal drops.* Instead, the ear and canal should be cleansed as far as possible with dry sterile cotton and then protected from germs from without. May we not draw from this some useful deductions as to our routine methods in the treatment of certain lesions?

In the line of major operations, we may mention that of Professor Moure for accidents of the mastoid or tympanic region resulting in facial paralysis. Having widely exposed the tympanic and mastoid fields, Moure makes a careful and systematic exposure of the facial nerve, beginning his uncovering of the Fallopian canal just over the fenestra ovalis and thence tracing the nerve backward and downward to its exit from the canal at the stylomastoid foramen. If the nerve is found intact, even though bruised, he is content with having removed pressure; if divided, the ends are sutured. If this very logical operation is possible in war time surgery, surely it should be more so in times of peace. And if successful, how obvious the advantages over the older operations for engrafting the peripheral segment of the injured facial into the sound trunk of another cranial nerve.

But valuable as the lessons in aural surgery may ultimately prove to be, there is another chapter of the medical history of the war in which otologists are more deeply interested, *i.e.*, that which deals with the various types of *war deafness.* These functional disturbances vary with the agency producing the injury, the character and site of the lesion, the position of the person at the time of the injury (*i.e.*, whether in the open or in a confined space, *e.g.*, trench) and finally with the morale, or mental and nervous organization, of the individual. Evidently there are certain injuries which by their destructive character must produce absolute or profound and permanent deafness. There are other injuries followed by a deafness equally profound, which eventuate in more or less rapid amelioration or recovery. And since in certain cases the deafness produced by these lesser injuries fails to exhibit the expected amelioration, even though the labyrinth may show in certain directions normal reactions, the conclusion is reached that under certain conditions a deafness originally induced by an insult to the perceptive labyrinth may be maintained or perpetuated through a psychic disorder. In other words, there is a type of war deafness which can be correctly interpreted only as a psychosis.

For various and obvious reasons what has been learned of the deafness resulting from near-by explosions as well as that resulting from direct cranial injuries by projectiles, bits of shrapnel, etc., has been based upon the clinical phenomena observed, and to a very limited extent only upon post-mortem studies.

According to Bourgeois, no organic lesions of the labyrinth have been observed or recorded as a result of injuries primarily involving only the soft parts, even in the case of so intimate a structure as the membranous auditory canal.

Of direct cranial injuries, those of the mastoid region naturally involve the labyrinth in the largest percentage of cases. According to the

statistics of Lannois and Chavanne,[1] in 38 cases of direct wounds of the mastoid region, total deafness resulted in 73 per cent. and partial deafness in 21 per cent. of cases. In the 23 cases constituting the statistics of Bourgeois, 60 per cent. and 33 per cent. resulted in total and partial deafness respectively. Next to the mastoid comes the temporo-maxillary region in the frequency of actual injury to the labyrinth, with deafness in 66 per cent. of cases. In half of these cases the deafness was absolute; in the reminder, partial. Of other parts of the skull, injuries of the occipital and temporo-parietal regions frequently involve the labyrinth (Chatelin). On the other hand, serious injury to the labyrinth as a result of military wounds of the frontal region is comparatively rare.

According to Bourgeois, the disturbances of hearing are more conspicuous than the disturbances of equilibrium in lesions of the labyrinth resulting from direct cranial injuries. This does not mean that the static labyrinths do not suffer, but that by comparison with the deafness, the disturbances of equilibrium often seem inconspicuous. Bourgeois states his belief that the deafness caused by a fracture involving the labyrinth is total and permanent. Naturally, these cases as a rule exhibit disturbances of equilibrium in the days immediately following the injury, but even in cases causing permanent and absolute deafness, he believes that the vertigo, subjective and objective, disappears wholly in time. This, of course, is in accordance with what we know of the sequence of events in other lesions of the labyrinth.

Fortunately, in the great majority of cranial wounds resulting in serious lesions of the labyrinth, only one labyrinth is involved, *i.e.*, that corresponding to the side of the cranial injury.

Of the actual pathology of these wounds, but little need be said since there has been little or no opportunity to check up clinical observations by properly conducted post-mortem examinations. When cranial injuries have been followed by profound and apparently permanent deafness, destructive changes in the labyrinth have been assumed, whereas a partial deafness or an immediately profound or absolute deafness showing more or less rapid or progressive amelioration naturally leads to a different conception of the nature of the labyrinthine lesion.

As to the pathogenesis of organic lesions of the labyrinth resulting from direct cranial wounds, different views are held by different observers. Le Mee and Toubert[2] believe that all organic labyrinthine lesions complicating cranial wounds are the result of gross or minute fractures of the labyrinthine capsule, produced either by direct violence upon the labyrinth or by extension from fractures of the base of the skull or from fractures extending downward from the temporo-parietal region. With this view Lannois, Chavanne and Bourgeois do not agree: recognizing fractures, gross or microscopic, of the labyrinthine capsule as occasional and possibly frequent war time lesions, these writers hold that rupture of minute blood vessels or tearing of terminal nerve filaments either of which might cause destructive or degenerative changes within the labyrinth, may be caused by

[1] Quoted by Bourgeois and Sourdille.
[2] Quoted by Bourgeois and Sourdille.

cranial injuries which produce absolutely no loss of continuity of the labyrinthine capsule. Considering the extreme delicacy of certain parts of the membranous labyrinth and the intense commotion and pressure to which they may be exposed by the severer cranial injuries, the latter view seems based on better logic.

From the foregoing statements, particularly as to the high percentages of absolute and permanent deafness, one's first impression is of a vast aggregate destruction of hearing in the late war. Deplorably great it probably has been. But we must remember that the percentages so far given have referred only to the cases of deafness due to direct cranial injury; and that such cases, when compared with those of deafness due to other and lesser causes, form so small a minority as almost to justify their being spoken of as among the rarer mishaps of the war.

Concussion Deafness Due to Explosions.—All observers are apparently agreed that great numbers of men suffered deafness of varying degrees and of various periods of duration as a result of air concussion, or the sudden and great displacement and condensation of air incident to near-by explosions. As regards this class of cases, certain facts apparently stand out. (1) While it is conceded that an aerial concussion from a near-by explosion or even under certain conditions from the firing of large cannon, may produce such violent disturbance of the cochlear structures as permanently to destroy their function, the consensus of opinion is that such cases are comparatively rare. Complete and permanent deafness is therefore exceptional. (2) Deafness due to concussion is usually unilateral, and confined to the ear nearest the explosive sound, *i.e.*, provided that at the time of the explosion the individual is in the open. It may be, and usually is, bilateral, when caused by an explosion while he is in a confined space, *e.g.*, dug-out or trench. (3) In cases of labyrinthine disturbance due to concussion from loud explosive noises, the cochlear mechanism is regularly more affected than the vestibular apparatus. As a rule the vestibular mechanism escapes wholly.

As throwing a considerable and very practical light on concussion deafness, the experiments of Prenant and Castex[3] are exceedingly interesting and instructive. These investigators subjected rabbits and guinea pigs to the detonations of heavy cannon of modern type, and then made careful observations of the labyrinthine changes induced. The following changes were noted: (a) dislocations of the ductus cochlearis, frequently with destructive changes in Corti's organ. (b) Effusion of blood in scala tympani due to rupture of minute vessels. (c) Atrophy of Organ of Corti and of all structures on cochlear surface of basilar membrane. (d) Apparent atrophy of ganglionic cells of the spiral ganglion. (e) (Rare) degenerative changes in central fibres of cochlear nerve. Perhaps the most important observation in the diagnostic light it throws on these cases is the negative finding, (f) "Integrity of the organs of the static sense noted in every case."

The pathologic findings in the experiments above quoted are extremely

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[3] Prenant and Castex, quoted by Bourgeois: War Otitis and Deafness, Pp. 89, 90.

important in that they supply the most clear-cut differential point between the labyrinthine lesions due to direct cranial injuries and those of the severest type (*i.e.*, causing absolute and permanent deafness) due to aerial concussion. If we admit that cranial injuries involving the labyrinth are likely to, or in fact almost of necessity must, involve both the cochlear and vestibular portions, and that the severest aerial concussions involve only the cochlear apparatus, it is clear that the ultimate clinical aspects of the severest types of these two lesions will be: (a) in the case of the labyrinthine lesion secondary to direct cranial injury, absolute deafness plus a non-irritable vestibular mechanism (*e.g.*, to cold or heat); and (b) in the case of a severe labyrinthine lesion from concussion, *deafness advanced or absolute, with retention of a normally functionating static labyrinth.*

The comparative immunity of the vestibular and semicircular canal system from serious injury by concussion, even following the loudest detonations, will become clear to us if we consider certain differences in the physiology of the two mechanisms of the labyrinth. The cochlear mechanism, specialized for reacting only to sound waves from without, is naturally subjected to a disturbance by any convulsion in the surrounding air in direct proportion to its violence. The fluids and structures of the vestibular mechanism, on the other hand, being *normally* uninfluenced by any agency from without, and reacting only to changes in the position of the head, are practically shielded from disturbances of the air induced by explosions. It is probable that the disturbances of equilibrium following explosions are induced either as a sympathetic phenomenon on account of its close association with the anterior (cochlear) labyrinth, or perhaps in certain cases from the individual's being thrown with more or less violence by the force of the explosion.

All grades of concussion deafness are said to have followed every heavy artillery engagement or action. They have varied from comparatively slight impairment to profound or absolute deafness. Naturally in a continued engagement both ears are subject to the same disturbance,—so that the milder cases are frequently bilateral. As a rule fairly rapid functional improvement has occurred in the milder cases, and even in cases of immediately profound or absolute deafness, beginning recovery of function has in many cases been an early phenomenon. Naturally in the severest injuries recoveries have been slower and ultimately only partial. But the cases of complete and permanent deafness from concussion appear to have been comparatively rare.

As to the character of the deafness, this, from all reports, is the same in kind whatever the character of the injury which has caused it,—*e.g.*, diminution or loss of hearing alike by air and bone conduction. When the deafness is not absolute, therefore, Rinnè may remain positive, though it is clear that an apparently negative Rinnè may be present through the conduction of sound waves through the cranial bones to the sound ear. Weber is referred to the sound ear. The upper limit of tone perception is markedly reduced. These changes characterize the deafness due either to concussion or to direct labyrinthine injury up to the point of total deafness, when all differential points, other than the degree of deafness itself, fail.

In cases of very extreme or absolute deafness, the chief reliance in the differential diagnosis between cases due to a direct injury of the labyrinth (*e.g.*, through fracture of the capsule) and those due to a cochlear degeneration from concussion (*e.g.*, following the explosion of a mine or large projectile) is apparently the loss or persistence of the normal vestibular reflexes, *e.g.*, reactions to heat or cold. The importance of a correct differentiation is not merely academic, since the prognosis even in extreme cases of concussion deafness is known to be infinitely more hopeful than in cases of deafness from wounds directly involving the skull.

Hysterical or Psychic Deafness: Pithiatism: Psychic Mutism: Simulation.—Hysterical deafness has been a convenient term in otology, a substitute for a confession of ignorance,—a term, that is to say, which calls up or conveys no mental picture of any definite aural disorder. Babinski and Froment have utilized the rich clinical material of the war as a means to retest the theory of auditory hysteria, and have more clearly defined its meaning, its limitations and its varying clinical aspects. Independent observations and investigations by Secard and Bellin, by Brindel, by Gault and by Bourgeois of various phases of so-called functional deafness have on the whole brought support to their conclusions. For hysteria Babinski has substituted a new and more descriptive word, *pithiatism*, a term derived from two Greek words and meaning *curable by persuasion*.

Pithiatism implies not only the possibility of cure by persuasion, but also the fact that the disorder may in some degree be called into being by suggestion. This does not mean that the origin of a pithiatic disorder is of necessity a hallucination or purely imaginary. Pithiatic deafness, for example, as we shall see, is at its inception a veritable deafness, the inevitable sequence of a shock to the perceptive labyrinth which could have had no other result. So far it has no hysterical or pithiatic character. But as the actual functional disability begins to subside, other factors, psychic in character, come into play and finally gain ascendence. To the extent to which they interfere with a normal recovery, the disorder then takes on a hysterical, or pithiatic, character.

There can be no question that the late war has created an ideal environment for the class of disorders under discussion. We are to picture to ourselves vast numbers of men of all social strata, of all degrees of intellectual poverty or attainment, of all grades of moral stamina or weakness: dragged suddenly from home and family ties, subjected for weeks or months to dispiriting monotony, to physical and mental tortures of heat or cold, of nerve-racking fatigue, of heart-piercing anxieties for unprotected dependents at home, of brooding doubts and fears as to unknown but impending ordeals. We must picture these men hurled suddenly into an inferno of carnage, a chaos of sights and sounds of such frightfulness and terror as men have never faced before,—and we have a fitting laboratory in which to observe and study the origin and incipient phases of pithiatism.

After the engagement our patient, with or without other physical injuries, suddenly realizes that he is profoundly deaf. He has seen men killed

or mangled before his eyes; he has heard of others made totally and permanently blind. The sudden loss of hearing, superadded to the depression of deadly physical fatigue, typifies in his mind the scope and gravity of the catastrophe which has overtaken him. Though he may accept his condition without complaint, he feels that his usefulness is permanently impaired. Since he is obviously incapacitated for receiving and carrying out orders, he is sent to a hospital for observation and treatment.

At the hospital or observation camp his hearing is tested and the pronounced character of his auditory defect verified; and a course of treatment planned. But whether the physician in charge has or has not been able to correctly interpret his condition, its treatment in reality must meet two quite distinct problems: (a) to bring about a restoration of the auditory function (and for this the element of time is probably the chief factor) and (b) to convince the patient of the progress of his recovery. As the days or weeks pass, the patient's general physical and nervous condition responds to rest and care, but deaf, and hopelessly deaf, he still believes himself to be.

One factor which is said in certain hospitals or observation camps to have fostered this hallucination in a patient in spite of returning function, has been the fact of other actually permanently deaf individuals associated with him in a common routine plan of treatment, their failure to show any favorable response impressing him with a hopeless prognostic view of his own condition. Such a patient may become a typical pithiatic. He honestly regards himself as hopelessly deaf and loses interest in his own recovery; nor is he likely to exhibit a more healthy mental state unless he comes under the care of a physician who understands his condition and how to deal with it.

While all classes of men are subject to pithiatic deafness, it is said that men of low mental status and of little education are particularly prone to become its victims.

Several phases of pithiatic deafness conspire to obscure the diagnosis for a physician unfamiliar with the condition. In the first place, even after actual auditory recovery is in progress, the hearing is still impaired. To hear and understand conversational speech, therefore, requires effort; and this effort the pithiatic, harboring the obsession of permanent deafness, will not make. Such is his lethargy that his responses to hearing tests do not fairly represent his actual auditory ability.

As to how far wilful, or intentional, simulation or malingering may play a part in any given case may be a difficult point to determine. It is at least apparent, however, that a man who has philosophised over his deafness as permanent and therefore to be accepted, may appreciate certain compensatory advantages,—*e.g.*, immunity from active military service, etc. How these two psychic factors may react upon each other, and which may preponderate, may call for keen analysis as well as a broad and tolerantly human viewpoint.

Bourgeois describes three types of pithiatic deafness: (a) deafmutism; (b) absolute deafness, and (c) partial, though pronounced, deafness. Deafmutism excites suspicion (1) from the fact that acquired total and perma-

nent bilateral deafness is exceedingly rare; and (2) even absolute deafness of recent development does not usually produce mutism. Absolute deafness and extreme hardness of hearing are not usually difficult to gauge, but an added element of either pithiatism or intentional simulation increases the difficulty.

One thing must be clear from what has been said, *i.e.*, that the honest sufferer from pithiatic deafness and the malingerer have this point of similarity—they both possess greater hearing power than their reactions to the older classical hearing tests demonstrate. Therefore, certain tests which are designed, or at least employed, to trap the malingerer may be equally essential and useful in establishing a correct diagnosis in the case of the pithiatic. The two tests upon which most reliance is placed are the *Lombard test* and the *cochleo-palpebral test* (*Gault*).

With *Lombard's test* most aurists are familiar. The pithiatic obsessed with the idea that one ear is absolutely deaf, or that his auditory function is lessened to a degree greater than the actual impairment, is tested as follows. A noise apparatus, preferably Barany's, is adjusted to the sound ear and set in action to accustom him to its sound. He is then asked to recite or, if he is not illiterate, to read aloud from a printed page. He is instructed not to stop reading when the noise instrument is placed in action. While he is reading aloud, the machinery of the noise apparatus, the earpiece of which occludes his sound ear, is started. If the hearing of the opposite, supposedly diseased, ear is impaired, he will immediately speak in louder tones; if it is absolutely deaf, he may actually shout. If he continues to read in an even tone or with only slight elevation, we know that the deafness or impairment is not real. The test is based on the fact that the normal individual depends upon his perception of his own voice sound for the regulation of its tone and intensity.

Gault's cochleo-palpebral test is a utilization of the fact that a normal person, if an unexpected sound is suddenly heard near either ear, will contract the orbicularis palpebrarum of the eye nearest the source of sound. The patient is seated, the sound ear is tightly closed with a finger, and an assistant of the examiner, standing behind the patient, suddenly produces some mechanical sound not loud enough to impress the closed (sound) ear. It can be appreciated, therefore, only by the ear under investigation. If heard, there will be a noticeable contraction, or winking movement, of the corresponding eye; and conversely from this winking movement, however slight, we have reliable evidence that the sound has been heard by the supposedly diseased ear.

These are the two tests which have apparently been most useful in the diagnosis of difficult cases of pithiatic deafness. Naturally if malingering is suspected, all the available tests for intentional simulation will be employed (see tests for malingering, page 96).

A complete statement of the routine methods of examination and of the treatment of these patients, even if the writer were competent to present it, the space at his disposal will not admit of here. That there are many perfectly honest pithiatics—*i.e.*, sufferers from psychic deafness in whose dis-

orders malingering plays absolutely no part—is proved by the statements of distinguished otologists as to the ready and remarkably rapid response of many of these unfortunates to skilful treatment. And this treatment is not by the brow-beating tactics which might logically, but in the writer's opinion never wisely, be utilized to convict an obvious malingerer. On the contrary, it calls for the use of tact in the effort, not to accuse, but by the employment of established methods of encouragement and suggestion to efface an obsession and thereby awaken hope. As illustrating the absence of harshness in the rationale of treatment, a single instance may suffice:—to a patient tested by Lombard's or Gault's method a positive reaction to either of these tests is presented not as evidence that he has deceived himself and his physicians, but as an encouraging sign of an intact perceptive mechanism,—a fortunate fact upon which both he and his physician may base their hope of ultimate improvement or recovery.

Stress is laid on the fact that routine use of ordinary local methods of treatment, *e.g.*, inflation, etc., is unwise for the double reason that (a) they may do harm and (b) lack of favorable results appreciable to the patient may react unfavorably upon him.

Occasional use of certain auditory exercises, care being taken to select sounds he is known to hear with comparative ease, is advised, not for any expected influence on his actual hearing power, but for their influence upon his morale, and with a view to stimulating his *will* to hear.

In conclusion the writer wishes to acknowledge his indebtedness to the admirable and interesting book, *War Otitis and War Deafness*, by Doctors Bourgeois and Sourdille, which has supplied most of the material for this chapter. If frequent references to it have been absent from his pages, that omission has been solely in the interest of brevity, and with this inclusive statement in mind.

For the convenience of readers, we append below a short list of papers bearing upon the subject matter which we have attempted in this chapter briefly to outline.

1. L'organe de l'audition, pendant le guerre: Labyrinthisme, examen fonctionnel. Prof. E. J. Moure et P. Pietri. Archives de Médicine et de pharmacie militaires. Vol. 65, Pp. 809-837.

2. L'organe de l'audition pendant le guerre: Le sourd cerebral, l'exagérateur, le simulateur. Prof. E. J. Moure et P. Pietri. Revue de laryngologie, d'otologie et de rhinologi, Vol. 38. Pp. 185-208.

3. Recherches expérimentales et histologiques sur la commotion des labyrinthes. Prof. Prenant et le Dr. A. Castex. Abstracted in Revue de Laryn, etc. Vol. 38. P. 427.

4. Paralysies faciales de la guerre. Prof. E. J. Moure; Presse Medicale, April 13, 1916.

5. Blessures du conduit auditif externe. Dr. J. Rozier. Revue de Laryng., d'otol. et de Rhin., vol. 38, Pp. 361-374: 384-400.

6. Etude sur les traumatismes de l'appareil auditif. Dr. H. Abrand. Revue de Laryn. etc. vol. 38. Pp. 433-447. 457-467.

7. Morbid Anatomy of War Injuries of the Ear: Drs. J. S. and John Fraser, Journal of Laryngology, Otology and Rhinology. Pp. 340-353. 369-385.

8. War Deafness with Special Reference to the Value of the Vestibular Tests: Drs. P. McBride and A. L. Turner: Lancet, July 20, 1918.

9. Effects of High Explosives on the Ear: J. Gordon Wilson, British Medical Journal, Mar. 17, 1917.

10. Further Report on the Effects of High Explosives on the Ear: J. Gordon Wilson: British Medical Journal. May 5, 1917.

11. Effects of High Explosives on the Ear: J. Gordon Wilson, N. Y. Medical Journal, February 23, 1918.

CHAPTER XXIII.

SALVARSAN IN THE TREATMENT OF AURAL DISEASE. AUDITORY NERVE LESIONS OCCASIONALLY FOLLOWING ITS USE.

Syphilitic lesions of the middle ear are in many cases more difficult of accurate diagnosis than in any other part of the body. It is evident that a specific ulcer at the pharyngeal mouth of the tube may spread through the canal to the tympanum. If a syphilitic ulcer within the tympanum spreads outward through the external auditory canal, there may result a destruction of tissue so characteristic as to suggest *per se* the etiologic basis of the lesion. Frequently, however, such extension does not take place, the lesion remaining confined within the tympanic spaces and assuming early the character of a chronic middle-ear suppuration of offensive and most intractable type. If the physician has failed to obtain a history of syphilitic infection and his suspicion has not been aroused by other manifestations of the disease, he may remain in ignorance of the character of the aural lesion. And yet the uselessness in such a case of any form of local treatment which is not combined with constitutional remedies must be obvious. The writer is inclined to believe that such negligence is not so rare as it should be.

As the tympanum, and in fact any part of the sound-conducting apparatus, may be attacked by the syphilitic virus, so also, though with less frequency, the auditory labyrinths or auditory nerves may become involved.

There is no question that the introduction of salvarsan has resulted in a world-wide renewal of interest in the study of syphilis in all its various phases. This has led not only to the establishment of certain facts in regard to the influence and value of the drug in question, but also to a clearer knowledge of many phases of the disease itself. For example, the analysis of a large series of cases compiled from the clinical records of institutions in various parts of the world has thrown a stronger light than has ever before been shed upon the cranial-nerve lesions occasionally resulting from syphilis. Seen thus in the aggregate, the number of such nerve lesions has reached a total sufficiently large to be impressive. Under this stimulus for further investigation, it is probable that in many cases cranial nerve disease has been observed and duly chronicled which formerly might have escaped notice, or at least might not have been recorded.

It has been found that certain cases of syphilis treated by salvarsan have developed cranial-nerve lesions, and that, of the nerves involved, the eighth pair has been more frequently attacked than any of the others. It has been stated further that under treatment by arsenobenzol ("606") a larger number of cranial-nerve lesions have occurred during the early

stages of syphilis than would appear from earlier records to have resulted from the older methods of treatment. Arguing from these premises, it has been assumed by some observers that the drug itself, rather than the syphilitic virus, has been responsible for the lesions of the optic and auditory nerves occurring during the early stage of the disease. Logical as this deduction would at first seem, it is one to which the facts, as they accumulate, seem less and less definitely to point.

Cranial-nerve Lesions following the Use of Salvarsan.—J. Benario[1] (Frankfort-am-Main) has collected from the literature 14,000 cases of syphilis treated by salvarsan, which he investigated with special reference to the frequency and type of cranial-nerve involvement. The results of his analysis, which are both interesting and instructive, may be stated as follows:

In the 14,000 cases the use of salvarsan was followed by severe disturbance of one or other of the cranial nerves in 126 cases, or 1 in every 111 cases. Of these cranial-nerve lesions the auditory nerve was most frequently involved (occurring in 62 cases), while the optic nerve (41 cases) came next in vulnerability.

Comparing the total number of auditory-nerve lesions following salvarsan injections (62 cases) with the total number of cases of salvarsan administration analyzed by Benario (14,000 cases), we find that the auditory nerve was involved once in every 226 cases treated. This percentage, if substantiated by further observations, is so small that it should not be allowed to weigh heavily against the enormous advantages which the drug in other respects seems to possess over the older remedies.

Pursuing his analysis yet further, Benario found that of the 126 cases of cranial-nerve lesions following salvarsan, 118 occurred in cases treated during the primary and early secondary stages, and only 8 in cases treated during the tertiary stage.

Of the 62 cases of auditory-nerve disease, 11 were accompanied by syphilitic involvement of other nerves. This leaves 51 cases in which the auditory nerves bore the brunt of the attack. Of these 51 cases, the cochlear branch alone was involved in 29, the vestibular branch alone in 5, and both branches in 17 cases.

In the 126 cases reported upon, evidences of a nerve lesion occurred in 96 per cent. of cases within 4 months from the administration of salvarsan, and 40 per cent. during the second month.

Ehrlich's explanation or theory in regard to the causation of these lesions is that they are due to the administration of salvarsan in insufficient doses,—*i.e.*, doses which have been large enough to control the general manifestations of the disease, but which have failed to devitalize certain isolated groups of spirochætæ lodged in the cranial-nerve sheaths; and that the activity of these neglected spirochætæ gave rise to a perineuritis.

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[1] Benario: Zur Statistik und Therapie der Neurorezidive unter Salvarsanbehandlung, Münch. med. Woch., vol. 58, No. 14, p. 732.

Benario, Desneux, and Dujardin all accept the conclusion of Ehrlich that these nerve lesions are properly to be regarded as resulting from the syphilitic poison, and not directly from the salvarsan administered. To the support of this view Benario brings the following facts:

1. The long interval frequently intervening between the injection of salvarsan and the manifestation of cranial-nerve involvement.

2. The nature of the pathological process itself, which, as is particularly evident in the lesions of the optic nerve, exhibits the characteristic features of an irritative or inflammatory lesion (Schantz, Tobias), rather than those of nerve atrophy, which is the lesion usually observed as a result of poisoning by other arsenical preparations.

3. That the disease (*i.e.*, nerve lesion) occurs almost exclusively during a fixed period of the disease.

4. That, so far as is yet known, such nerve lesions are not observed in diseases non-syphilitic in origin, following treatment by salvarsan.

5. The curability of such nerve lesions by antisyphilitic remedies, and particularly by salvarsan injections.

6. That such nerve lesions have usually followed the administration of small doses of salvarsan.

7. That exactly the same manifestations have occurred under treatment by mercury.

Benario believes that the exciting cause is in certain cases to be found not in the chemical action of the drug, but rather in failure to maintain complete sterility (*i.e.*, employment of a solution not wholly sterile).

Dr. Duel, of New York, who carefully reviewed the literature of the subject up to 1911, contributed to the volume of "Progressive Medicine" for that year a short but instructive *résumé* of the work so far accomplished. As to the deductions to be drawn therefrom, Duel believes, with the authors above cited, that the auditory-nerve lesions following the administration of "606" are usually to be interpreted as manifestations of the disease, and not as accidents resulting from the injections of arsenobenzol.

As contrasting with the above conclusions, the views of Prof. Alexander,[2] of Vienna, are of interest. Alexander's contribution to the subject is based upon a careful clinical study of cases referred for aural examination from the dermatological clinics of the Vienna General Hospital and the Vienna Polyclinic Hospital prior to 1910. During this time he had observed 68 cases of syphilitic disease of the ear. Only 9 of these had been indubitable cases of syphilitic involvement of the auditory nerve occurring in the early stages of syphilis. Of these 9 cases, 4 were of very slight and transient involvement, all symptoms rapidly disappearing under mercurial treatment. This left therefore only 5 cases of serious syphilitic involvement of the auditory nerve occurring in the early stages of the disease during a period of six years. Yet Prof. Finger had seen 3 such cases within

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[2] Alexander: Possible Effects upon the Auditory Labyrinth of the Ehrlich-Hata Remedy, Annals of Otology, vol. xx, No. 2, pp. 441–9.

a period of six months. The 3 cases of Prof. Finger had been of patients treated by salvarsan. The 5 cases of Alexander were of patients treated by the older method,—*i.e.*, mercurial inunctions, etc. Alexander, therefore, concludes that the increased frequency of auditory-nerve involvement as shown in Finger's cases is in some way related etiologically to the use of salvarsan. Referring to the previous literature, Alexander calls attention to the great rarity of syphilitic disease of the auditory nerve during the early stages of the disease prior to the introduction of salvarsan.

Summing up his personal deductions, Alexander believes (1) that, when acute or chronic disease of the auditory nerve or labyrinth is present in the early stages of syphilis,—whether the auditory-nerve lesion be syphilitic or non-syphilitic,—salvarsan is a dangerous drug and should not be administered; (2) that in the tertiary stage of syphilis or chronic or latent syphilis, the occurrence of an acute affection of the auditory nerve should be regarded as a contra-indication to the use of salvarsan; (3) on the other hand, there are a large number of cases of chronic syphilitic involvement of the auditory nerve and chronic labyrinthitis complicating chronic syphilis in which he regards salvarsan as a safe and valuable remedy.

The above conclusions are of interest and are worthy of careful consideration. They can not, however, be accepted as reliable guides to treatment, for the reason that the interpretation of clinical facts, upon which they are based, is open to question.

For example, Alexander's views are based upon his belief that the occurrence of cranial-nerve lesions during the early stages of syphilis is a development dating chiefly from the introduction of salvarsan. Even should this fact be susceptible of proof,—which apparently it is not,—further investigation would still be necessary in order to determine whether the increased frequency of such "neuro-recurrences" is due to the chemical action of the drug itself or to some factor depending upon a faulty method of administration.

Benario[3] has analyzed 29 cases of cranial-nerve lesion in syphilis treated by injections of mercury. Ten of these were lesions of the auditory nerves, in all of which the evidences of nerve involvement followed the mercurial injections. Of this series of 10 cases, in 8 the deafness following mercurial injections was relieved by injections of salvarsan. The restoration of function following salvarsan injections was partial in two cases, complete in six cases. In one case the symptoms of auditory-nerve involvement disappeared under further mercurial treatment. In the remaining case the deafness following treatment by mercurial injections was not relieved by subsequent injections of arsenobenzol. It would seem, therefore, that cases of syphilis treated by injections of mercury and cases treated by injections of salvarsan are alike subject to cranial-nerve lesions.

[3] Benario: Ueber syphilitische Neurorezidive in besondere solche nach Quecksilberbehandlung, Münch. med. Woch., vol. 58, No. 1, p. 20.

Fordyce believes that, when in any considerable series of cases treated by salvarsan the number of cranial-nerve lesions reaches an unusually high percentage, the possibility of a faulty technic should be considered. In support of this view, he calls attention to the fact that these nerve lesions were far more common during the earlier experiences with the drug, —*i.e.*, before the importance of using only freshly prepared solutions, made with freshly distilled water, was appreciated, and during the time when the drug was administered in doses now generally recognized as having been insufficient.

Referring to Professor Finger's report as to the frequency of cranial-nerve lesions following the administration of salvarsan, a comparison of his reported ratio with that of others, cited below, furnishes food for thought:

Finger, Vienna	500 cases; cranial-nerve lesions, 44
Wechselmann, Berlin	2800 cases; cranial-nerve lesions, 10
Arning, Hamburg	1000 cases; cranial-nerve lesions, 2
Schreiber, Heidelberg	1060 cases; cranial-nerve lesions, 1
Fordyce, New York	800 cases; cranial-nerve lesions, 1

In the light of Yakimouf's experiments, proving that salvarsan solutions may carry active pathogenic bacteria, and Ehrlich's warning as to the necessity of using only fresh solutions of the drug made with freshly distilled water, so disproportionately large a percentage of cranial-nerve lesions as are reported by Professor Finger, forces one to consider the possibility of some error either in his method of preparing the solution or in the method of its administration.

The Use of Salvarsan.—The most optimistic view at any time held as to the possibilities of salvarsan was that by its use we might in favorable cases be able to destroy all the spirochætæ in the infected subject by a single dose. To render this possible, even in the most favorable phase of the disease, it is necessary that the drug be administered in large doses and intravenously. Small doses, failing to devitalize all the germs, do not terminate the disease, and, therefore, pave the way for recurrence of symptoms. Interstitial injections are so slowly absorbed that the full strength of the dose does not impress the organism at the same moment, the gradual absorption not only preventing the simultaneous neutralization of all the spirochætæ in the body, but having the further disadvantage that it allows time for the gradual development of immunity on the part of those spirochætæ which escape the early influence of the drug, so that subsequent doses, even though large, are less effective in eliminating the disease.

While complete elimination of the poison as the result of a single dose remains a possibility, this happy result is so seldom attained that we have learned through experience a more rational use of the remedy. Since the so-called intensive plan of treatment has been introduced, and particularly since the wisdom of combining the use of salvarsan with that of mercury has gained recognition, the results have been far more satisfactory.

As to the practical application of salvarsan to the cure of syphilis, a condensed statement of some of the conclusions of Dr. John A. Fordyce, than whom probably no man in America has had a larger experience, should be of value. His plan of administration is to give first what he now regards as a moderate dose,—*i.e.*, gm. 0.3 to a woman or gm. 0.4 to a man,—to test the patient's tolerance of, or idiosyncrasy against, the drug. No contra-indications arising, this is followed within one week by a second injection of gm. 0.4 or gm. 0.5, according to sex. It is generally advisable in early cases of the infection, if no contra-indications are present, to give from five to six injections of salvarsan at weekly intervals followed by a course of mercurial injections or inunctions. If, after a month or six weeks of rest, the Wassermann reaction continues positive the same procedure may be repeated.

Summing up his observations of therapeutic results, Fordyce says: "Since the adoption of an intensive and combined plan of treatment, I have noted no relapses whatever in about 200 patients treated in the early stage of syphilis."

As to the cranial-nerve lesions following the administration of salvarsan, Fordyce states that in a series of between 800 and 900 cases there has developed not a single lesion of the auditory nerve and only one of the optic nerve. In this case unilateral optic neuritis occurred in the early secondary stage, and six weeks after the administration of salvarsan.

As to the curative influence of salvarsan upon pre-existing lesions of the optic nerve, Fordyce states his belief that it neither cures nor accelerates optic atrophy, but that it does exert a distinctly favorable influence upon optic neuritis *coming on during the early period of syphilis.* If this observation is correct, there is no apparent reason why the same may not be true of lesions of the auditory nerve.

The great importance of carefully sifting the facts, and giving to contradictory theories their proper values, is made clear by a comparison of the following views: Alexander, of Vienna, from his observations of aural lesions following the administration of salvarsan, concludes that salvarsan should not be given in the early stages of syphilis in the presence of a labyrinthine disorder of any grade or type. Fordyce, from his observation of the influence of the drug in clearing the system of the syphilitic poison, states his belief that it is in the early stages of the disease that the drug is most surely curative. Shall we deprive the patient of the advantages of salvarsan during the stage of the disease presumably most susceptible to its influence on account of a doubtful hypothesis that its use predisposes the patient to syphilitic involvement of one or more of the cranial nerves?

CHAPTER XXIV.

VACCINE THERAPY IN THE TREATMENT OF AURAL DISEASE: AUTOGENOUS VACCINES; BACTERICIDAL SERA

THAT serum treatment in some form will continue to play an important rôle in the control of disease, there can be no doubt. While its field of usefulness in certain forms of aural disease has been made the subject of many interesting papers, it cannot be said that the clinical experiences of different investigators or in different institutions have been sufficiently uniform to bring universal recognition of its value. There is need of further investigation in order to determine more definitely its field of usefulness and its limitations.

In venturing to touch upon this important subject the author will not attempt more than to state briefly the rationale of the remedies under consideration, and to indicate certain types of aural disease to the relief of which they have seemed to contribute.

PRELIMINARY REMARKS.—In the first place, it must be remembered that the pneumococci, streptococci, staphylococci, and other germs usually associated with aural suppuration are of the class of bacteria which have the property of elaborating within themselves certain toxic substances known as endotoxins. The endotoxins of this class of bacteria are not yielded readily to the circulating fluids of the body, being set free only with the death or approaching dissolution of the parent micro-organism. Each particular germ produces only its own specific endotoxin, which differs from all the others, and is neutralized only by antibodies called into being by the parent germ. This is an important fact bearing particularly upon the therapeutic value of autogenous vaccines, and upon the frequent worthlessness of stock vaccines.

The bacteria of aural suppuration do not, then, elaborate an easily soluble toxin which reaches the circulating fluids during their period of life; and they exert their morbid influences chiefly in two ways,—viz., (1) by the irritation caused by their physical presence within the tissues; and (2) by the systemic disturbances caused by the circulation in the blood of the endotoxins which are set free by the death of each succeeding set of bacteria.

Before considering the remedies at our disposal, let us recall briefly some of Nature's weapons of defence against such bacterial invasion,—viz., the phagocytes, opsonins, and antibodies.

I. The *leucocytes*—or, as they are commonly called in their relation to the control of disease, the *phagocytes*—have the power of actually destroying pathogenic bacteria. Under conditions of normal resistance, the development of a suppurative lesion in any part of the body is followed

by a migration of leucocytes to the part and, if the lesion is of sufficient severity to threaten systemic disturbance, by an increase in their total number circulating in the blood. Any increase in the number or virulence of the invading germs is accompanied or followed by an increase in the number of leucocytes, and particularly of the polymorphonuclear cells. If, however, the bacteria gain the ascendency, the continued call for reinforcements may exhaust the resources of the organism, and we shall have the spectacle of an individual becoming progressively more septic; while examination of the blood shows a diminishing leucocyte count and a diminishing polymorphonuclear cell percentage. These, of course, are generally recognized clinical facts. The clinical course of the disease may, therefore, be regarded as to some extent a record of the changing balance of power between the invading bacteria and the phagocytes.

II. *Opsonins.* — There exist normally in the blood certain bodies, called opsonins, which can not themselves destroy the bacteria, but which have the power of reducing their resistance, thereby rendering them vulnerable to the attack of the phagocytes. In this way they increase the effective activity of the phagocytes.

III. *Antibodies.* — In any suppurative lesion the presence of the bacteria within the tissues not only influences a migration of leucocytes to the part, but has also the effect of stimulating the organism to the production of certain substances known as antibodies. These antibodies support the phagocytes in that they either destroy or neutralize the germ by whose presence they have been called into existence. A fact which must be borne in mind is that each pus-producing micro-organism elaborates its own specific endotoxin and excites or stimulates the organism to the production of its own specific antibody. In other words, the antibody called into being by any particular germ will effectively destroy or neutralize only that particular germ and its specific endotoxin.

With even so brief a statement of certain of Nature's means of combating infection, we are better able to appreciate man's attempt to turn her weapons into therapeutic agents under his control.

Autogenous Vaccines.—The vaccine treatment of a suppurative lesion is not an attempt to introduce into the system substances inimical to the bacteria, but rather an effort to stimulate the organism to the production of antibodies corresponding to the micro-organisms present. An autogenous vaccine is prepared by cultivating the bacteria of the infection from pus obtained from the lesion. The bacteria thus cultivated are killed by subjection to a temperature of 60° C., the devitalized bacteria being suspended or dissolved in normal salt solution. It is found that this solution, injected interstitially, subjects the patient to no risk of further infection, but retains the power of stimulating the organism to the elaboration of the corresponding antibodies. Naturally, if we can incite the body to a production of antibodies potentially overbalancing the bacteria present, a practical immunity will be established.

If we are to make intelligent use of vaccines in the control of suppurative lesions and to interpret correctly their results, we must bear in mind the following facts:

1. The vaccine supplies nothing to the organism wherewith to destroy the bacteria present or to neutralize the endotoxins in the blood. It incites the organism to an increased production of antibodies and possibly of opsonins. The results, measured by clinical changes for the better, can not, therefore, be otherwise than gradual. Whoever expects immediate amelioration of symptoms is doomed to disappointment.

2. The use of vaccines actually places a greater tax upon the system. During the interval, therefore, between the first injections of the serum and the establishment of an increased production of antibodies, the condition of the patient may seem—and, for the time, actually be—less favorable.

3. During the height of a severe acute suppurative lesion, the organism may be taxed nearly to the limit of its resources. Under such conditions, the use of a vaccine might disturb the balance of power disastrously, producing such systemic exhaustion as to leave the bacterial poisons unopposed.

4. For reasons deducible from the facts stated in the foregoing paragraph, the use of a vaccine in cases of profound sepsis—*i.e.*, in which the powers of resistance are already exhausted—may further deplete the patient's vitality, with disastrous consequences.

Autogenous versus "Stock" Vaccines.—The preparation of an autogenous vaccine represents a scientific attempt to isolate from the germ causing the infection that part of it which stimulates the organism to the production of the appropriate antibodies. Its use is, therefore, an application of the principle of cause and effect.

The stock vaccine is a similar preparation from some typical pathogenic bacterium,—*e.g.*, a streptococcus or staphylococcus,—or from several strains of bacteria belonging to the same general type. Its employment ignores the important fact that germs of the same general type may differ materially in virulence and probably in other characteristics, that they elaborate endotoxins of different toxicity and call into being antibodies exhibiting like variation. If we admit as a working hypothesis that the antibodies called for by a streptococcus of certain grade or type are powerless to destroy or neutralize a streptococcus of a different grade, we should be at no loss to explain the frequent failure following the use of a stock vaccine. If the germ from which the stock vaccine was prepared happens in all respects to coincide with the micro-organism causing the infection, the result should be favorable. If we employ a stock vaccine made from a germ which is not coequal or coöperative with that of the lesion, its employment will not only result in failure, but will actually deplete the patient by placing an additional tax upon his resources,—*i.e.*, by calling for the production of antibodies which are quite ineffective against the bacteria and endotoxins present.

The aural lesions which have responded most favorably to treatment by autogenous vaccines are the subacute and chronic types of infection, or those tending toward chronicity. Among these may be mentioned (a) subacute suppurative otitis media,—*i.e.*, certain cases of acute purulent otitis media which, having passed the acute stage, obstinately resist the usual methods of local treatment; (b) furunculosis of the canal, the use of the autogenous vaccine establishing an active immunity which prevents the recurrences which are so characteristic of the lesion; (c) probably some operative cases,—*e.g.*, following the radical operation,—in which a low form of tubal infection delays recovery. The above lesions, it will be recalled, are not those which are likely to tax to the limit the vitality and resources of the organism. They provide, therefore, an ideal condition for the establishment of an active immunity. Corroborating this view is the fact that the lesions mentioned are those in which the results of the vaccine treatment have been most satisfactory.

Bactericidal Sera.—The use of bactericidal sera—*i.e.*, the sera of animals which have been immunized against the bacteria corresponding to the infection to be controlled—has not, so far as the writer knows, been shown to have any wide or well-defined field of usefulness in aural disease. Their value and place in aural therapy must, therefore, be considered disproved, or await further investigation.

CHAPTER XXV.

AURAL DISTURBANCES DUE TO DENTAL LESIONS.

It is clear that cases properly belonging to this category are not to be regarded as cases of aural disease. They may, therefore, be dealt with briefly.

That individuals whose hearing is perfect and whose ears exhibit structurally no abnormality may suffer acutely with aural symptoms, reflexly induced by lesions in other parts of the body, is a fact well known to aurists. Thus, acute tonsillitis or cancer of the base of the tongue may give rise to a severe grade of earache. By far the most common cause of reflex otalgia, however, is dental caries. This fact is so generally appreciated by aurists that it is a routine practice in every aural clinic to examine the teeth of patients complaining of ear pain whose ears do not show physical evidences of acute inflammatory disease. Very frequently the cause of the otalgia is clearly seen in teeth, or roots of teeth, in advanced stages of necrosis. In such cases the aurist is able to assure the patient that his suffering is not the result of aural disease, and that it will in all probability be relieved as soon as the carious roots have been removed or the teeth put in order.

There are many cases, however, which are not so easily diagnosticated as the type above described. For example, the patient may complain of aural pain, the ears may show no physical signs of acute disease, the throat may appear normal, and the teeth may exhibit no evidences of disease which the physician is able to recognize. In such a case one may consider the possibility of a neuralgic or rheumatic manifestation localized in the ear, but such a diagnosis should not be finally accepted until the teeth have been examined by a competent dentist.

A class of cases which until recent years defied correct interpretation is found in patients whose aural symptoms are dependent upon dental irregularities which even the dentist might fail to detect. These patients may suffer ear pain or may experience aural symptoms of less unbearable type. For example, some of the most perplexing and intractable cases of tinnitus aurium are now known to be the result of dental lesions or abnormalities the presence of which can not be determined except through the medium of a radiogram. I am inclined to believe, therefore, that every obscure functional disturbance of the ear—*i.e.*, in which no rational working hypothesis can be arrived at—should be referred to a competent dentist with the request that an X-ray picture be secured.

I am indebted to Dr. M. I. Schamberg, of New York, for the accompanying radiograms showing dental lesions of patients, some of whom were referred to him on account of symptoms referred to the ear. Fig. 329 shows graphically an abscess at the roots of two neighboring incisor teeth. In Fig. 330 the roots of a bicuspid tooth are seen projecting into the

antrum of Highmore. Fig. 331 represents the lower jaw of a patient who suffered from inflammation of the alveolar process and gum as a result of the imperfect (partial) eruption of a wisdom tooth. Figs. 332 and 333

Fig. 329.—Radiogram showing abscess at roots of two incisor teeth.

Fig. 330.—Radiogram: roots of a bicuspid tooth projecting into antrum of Highmore.

Fig. 331.—Radiogram showing imperfect (partial) eruption of wisdom tooth.

Fig. 332.—Radiogram: unerupted and abnormally placed tooth.

Fig. 333. — Radiogram: unerupted and abnormally placed tooth.

show unerupted and abnormally placed teeth. It is obvious that some of these lesions might be absolutely impossible of diagnosis except through the aid of a radiologist.

The purpose of this very short chapter is to place proper emphasis on the two types of cases referred to,—viz., (1) those in which severe earache is clearly attributable to a dental lesion the presence of which is easily recognized; and (2) those in which earache or more indefinite symptoms are the result of dental abnormalities not determinable by direct inspection. The latter are the more important, for the reason that, unless a correct diagnosis is made by means of a radiogram, the patient's symptoms are not likely to be relieved; and, further, he may be subjected to a prolonged course of treatment the results of which can not be beneficial and may be actually harmful.

CHAPTER XXVI.

DEAF-MUTISM.

THE TERM deaf-mutism is properly applied only to the condition of individuals who, by reason either of congenital deafness or extreme deafness acquired in the first years of life, fail to acquire or retain the power of articulate speech. According to these differences of origin, deaf-mutism is spoken of as congenital or acquired.

ETIOLOGY.—Of the causes of congenital deaf-mutism but little is known beyond certain general facts relating to heredity. Owing to the frequent segregation of deaf-mute children in schools and institutions and the friendships there formed, and their clannishness in adult life, intermarriage between deaf-mutes is exceedingly common. As to the influence of such unions in determining the birth of congenitally deaf children, the evidence furnished by statistics is less definite than one would expect to find it. While acquired deaf-mutism is rarely if ever transmitted, marriage between congenital deaf-mutes accentuates an inherited tendency, and the defect is more than likely to reappear in one or more of the children or grandchildren. On the other hand, an analysis of the family histories of the children attending the various institutions for the deaf and dumb shows that the percentage of those born of deaf-mute parents is much smaller than would be expected. In spite, however, of certain apparent contradictions, the evidence, as it accumulates, points more and more directly to the danger of such unions. As an extreme illustration of such transmission may be mentioned the case reported by Hartmann, in which a marriage between two congenital deaf-mutes resulted in the birth of four deaf-mute daughters and one normally hearing son. This, however, is a comparatively rare and extreme instance.

Better illustrating the average frequency of direct transmission are the statistics collected by Mygge, and analyzed by Mygind, of Copenhagen.[1] Mygge collected from the records of different countries the histories of 367 marriages between individuals one or both of whom were deaf-mutes, and found that of the children born to them 22 were congenital deaf-mutes,—*i. e.*, one deaf-mute child in every sixteenth or seventeenth marriage. Separating these marriages further into those in which both of the contracting parties were deaf-mutes and those in which only one was so afflicted, it was found that one deaf-mute child was born in every sixth or seventh marriage between two congenital deaf-mutes, whereas only in every thirtieth or thirty-first family in which one parent only was a deaf-mute was a deaf-mute child among the offspring. Naturally, taking all the children born of these 367 marriages, the proportion of deaf-mute

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[1] Mygind: Deaf-mutism, p. 46.

children to those of normal hearing was much smaller, the ratio in this series being as 1 to 57 or 58.

Many deaf-mutes—particularly those in whom the defect is directly traceable to diseases contracted after birth—are entirely without any history upon which an hereditary taint could be based; and the intermarriage of two such individuals entails little if any risk of the acquired defect being transmitted to the children. On the other hand, congenital deaf-mutes whose family records include the history of many deaf and deaf-mute members, by intermarriage assume for their children a frightful risk of inherited calamity. In other words, the consensus of opinion among those who have investigated this subject most thoroughly (Mygind, Love, Bell, Hartmann) is that an inherited tendency toward deafness on the part of the parents carries with it greater danger for the children than would the severest type of acquired deafness. According to this hypothesis,—which is in accord with the general theory of heredity,—a marriage between two normally hearing individuals in both of whose families many deaf-mutes had been born, would be far more likely to produce deaf-mute children than would a union between two deaf-mutes whose deafness had resulted from postnatal disease.

In investigating the family histories of deaf-mute children in Glasgow institutions for the deaf and dumb, Love came upon three families in which both father and mother were congenitally deaf. Among the children born to these three unions were five congenital deaf-mutes. Hartmann (quoted by Love) in Berlin came across only two families in which both parents were deaf-mutes. In one of these the parents were congenital deaf-mutes, and of five children born to them four were deaf-mutes. The other marriage was between individuals whose deaf-mutism had been acquired through disease contracted after birth, and their children were of normal hearing. Here, then, we have a record of five marriages between deaf-mutes,—four congenital and giving birth collectively to nine congenital deaf-mutes, and one between victims of acquired deaf-mutism to whom only normally hearing children were born.

As further emphasizing the influence of heredity, Dr. Love cites the Forty-seventh Report of the Cambrian Institute, in which is given a record of thirty-four families in which 130 children were born, of which number 54 were deaf. Such a record can only be explained upon a basis of heredity.

Relation of Parental Consanguinity to the Spread of Deaf-mutism.—There seems to be no question that a somewhat larger percentage of deaf-mute children are born to marriages between blood relations than to married couples who have no blood relationship. Beyond this fact, there is no evidence that consanguineous marriage between absolutely normal individuals influences the birth of deaf-mute children. And yet there is little doubt that absolute and effective prevention of so-called "cousin marriages" would tend in some degree to lower the birth-rate of deaf-mutes. Dr. J. K. Love has collected a large amount of interesting fact bearing upon the relation of parental consanguinity to the spread of deaf-

mutism, among which may be mentioned the following: Among the Roman Catholics of Germany whose church prohibits intermarriage between near blood relations, the proportion of deaf-mutes to the rest of the Catholic population is 1 in 3000. With Protestants, by whom intermarriage between cousins is not condemned and among whom it is by no means uncommon, the proportion is 1 to 2000; "while among the Jews, who encourage intermarriage between blood relations, the deaf-mutes are as 1 to 400" (Hutchinson).

Among authentic instances of isolated communities in which intermarriage has flourished is mentioned the Da Souza slave colony in the African kingdom of Dahomey, where several generations of almost promiscuous intermarriage or relationship had resulted in a progeny practically free from deaf-mutism or physical blemish. An even more striking example is the island of St. Kilda, situated far out in the Atlantic Ocean west from the Hebrides Islands, from which group it is as far distant as the latter are from the west coast of Scotland. The people of this island, practically cut off from active association with the rest of the world, have intermarried for centuries, and yet are free from deaf-mutism and other blemishes which such inbreeding might be expected to produce.

In contrast with these instances is the account given by Prof. Graham Bell in 1880 of the hamlet of Chilmark on the island of Martha's Vineyard, whose population had had little commerce with the people of the mainland, and whose families had intermarried for many generations. Among its population were 72 congenital deaf-mutes.

Apparently the influence of consanguineous marriage upon the normality or abnormality of the children is dependent upon the following conditions: In a marriage between cousins, should each be physically and mentally normal and free from hereditary taint, there should be little reason to expect other than normal children; if, however, there is in their family some inherited tendency toward a particular disease or physical defect,—*e.g.*, phthisis, insanity, polydactylism, deaf-mutism, or what not,—this inherited tendency will be intensified by their blood relationship, and the danger of its transmission to future generations is enormously increased.

The causes of acquired deaf-mutism fall necessarily under two heads,—viz., lesions attacking the auditory nerve from the intracranial side (meningitis), and suppurative processes reaching the labyrinth by way of the tympanum.

In the order of their frequency as causes of deaf-mutism may be mentioned the following diseases: cerebrospinal meningitis, scarlatina, measles, diphtheria, typhoid fever, parotitis.

Judging by the proportionate frequency of these various causes in the cases of acquired deaf-mutism applying for diagnosis and advice to the Manhattan Eye, Ear, and Throat Hospital of New York, there would seem to be no question that in this country more cases are the result of cerebrospinal meningitis than are due to any other cause.

According to Love's investigations it would seem that meningitis plays a smaller rôle in Scotland and the British Isles generally than in this country or in Continental Europe. From his collected statistics of European countries, one is led to believe that about one-third of all cases are due to meningitis, one-third to scarlatina and measles, and the remainder to various diseases, including typhoid fever, diphtheria, parotitis, etc.

While reliable statistics are not available in support of this contention, I am decidedly of the opinion that in New York at least one-half of all cases of acquired deaf-mutism are due to cerebrospinal meningitis. I base this belief upon my personal experience during the past ten years as chief of a large aural clinic in which a majority of the cases brought for advice on account of acquired deaf-mutism have been traceable to cerebrospinal meningitis.

The larger percentage of cases resulting from scarlatina and measles as compared with those due to other infectious diseases is explained by the fact—now generally recognized by those who have made a study of the aural complications of the acute exanthemata—that scarlet fever and measles more frequently give rise to severe forms of tympanic and mastoid suppuration than do all other infectious diseases combined.

Parotitis (mumps) is a comparatively rare cause of acute aural disease, but the occasional lesions from this cause are exceedingly prone to involve the labyrinth and to eventuate in extreme grades of deafness—a deafness, which, as a rule, is permanent.

Morbid Anatomy.—But little is learned of the pathologic basis of deaf-mutism from a study of living patients. It is not always possible by a physical examination of the ear to determine even whether the lesion is of congenital or acquired origin. Naturally, if it can be determined that the patient has never heard or spoken, and the ears present no physical abnormality, the inference of congenital deafness would seem justifiable. Congenital deaf-mutes are not, however, immune from tympanic disease, and the early development of a suppurative lesion has in many recorded cases left the origin of the deafness in doubt. Our real knowledge must, therefore, depend upon such postmortem findings as have been recorded and the inferences which are justifiable therefrom.

The following lesions are mentioned as having been actually observed in cases of deaf-mutism coming to autopsy: (1) Bilateral absence of the external auditory meatus (Hartmann). It is known that this condition is not infrequently accompanied by anatomical defects of the labyrinth. (2) Absence of the modiolus, lamina spiralis, and essential structures of the membranous labyrinth in the outer (*i.e.*, apical) half or two-thirds of the cochlear tube. Several variations of this defect were found in postmortem examinations upon congenital deaf-mutes by Ibsen and Mackeprang, of Denmark. (3) Abnormally narrow internal auditory meatus; rudimentary cochlea (Romer). (4) Complete absence of both labyrinths and both auditory nerves (Dardel). (5) Osseous closure of

both round windows; large quantity of otoliths and colloid corpuscles in both saccules and in both basilar membranes (Moos). (6) Bilateral absence of organs of Corti with atrophy of eighth nerves (Baratoux). The above records of actual postmortem findings are given in Mygind's classical work on deaf-mutism, which I acknowledge as the source of these references. Those wishing to examine the original records of these and other cases of deaf-mutism coming to autopsy are referred to the admirable bibliography contained in Mygind's book.

A recent contribution to this subject is the very comprehensive review of the literature relating to deaf-mutism, correlated with the results of his own investigations, by Dr. J. S. Fraser[2] of Edinburgh, Scotland. Dr. Fraser holds that the terms "congenital" and "acquired" as commonly applied to deaf-mutism may be misleading, since even total deafness at birth may, as when due to intra-uterine meningitis, belong more properly to the category of acquired lesions. He therefore prefers to divide all cases into (1) those due to a developmental error or defect (which necessarily must be in every sense congenital); and (2) those due to concomital disease, *e.g.*, meningitis, syphilis, infection, etc., whether developed in utro or after birth. While it is clear that there are many cases in which such a differentiation might be difficult or impossible, it is a suggestion which may well form the basis of useful and productive investigation.

Dr. Fraser gives two case histories with postmortem findings the salient features of which are here included in condensed form. Case 1. A boy, *æt.* 13 years, was practically a deaf-mute from birth, although he apparently could hear tuning forks of 256 double vibration and higher up to the limit of the Hartmann set in both ears. The right drum-membrane was intact, while in the left ear, the drumhead was partially destroyed and foul pus escaped from a chronically infected tympanum. As a result of a later extension from this focus, the patient developed a purulent leptomeningitis from which he died. The autopsy findings are here recorded only as they involve the labyrinths and chiefly as to cochlear changes.

Left Ear.—Neuro-epithelium of utricle normal but otolith membrane separated therefrom. Saccule collapsed and with epithelium normal only in parts. Duct of utricle abnormally dilated; that of saccule narrow. There was present a "considerable quantity of curdled lymph in the scala tympani." Corti's organ "is badly formed and nowhere is the normal acoustic papilla visible." Membrana tectoria nowhere normal and showing in various parts of the coil abnormal adhesions or contacts. In contrast to all these abnormalities, the acoustic nerve fibres wherever present appeared normal and the ganglion cells were present in normal number.

Right Ear.—Presented very similar changes. Collapsed saccule. Cochlea duct narrow and in parts collapsed. Corti's organ "is for the most part an irregular heap of cells." "Spiral ganglion cells appear normal in num-

[2] J. S. Fraser: Pathological and Clinical Aspects of Deaf-mutism; Journal of Laryng. and Otol., 1922, vol. XXXVII, pp. 13 to 38; 57 to 75; 126 to 137.

ber and the hollow space of the bony spiral lamina is well filled with nerve fibres."

Here clearly was a case of congenital deaf-mutism since no acquired disease could have produced the changes noted.

Case II.—This is described as a case of "acquired or inflammatory deaf-mutism." The patient, a boy of ten years, was one of twelve children, none of the others being deaf. He had never spoken.

Examination: In the right ear there was a perforation in Shrapnel's membrane ("attic perforation") through which cholesteatoma could be seen, and pus escaped into the canal. The left drum-membrane was intact, though exhibiting an "adherent scar" in the upper-posterior quadrant.

Six months after the examination recorded above, the patient developed a right-sided mastoiditis complicated by infective sinus thrombosis, from which, in spite of jugular vein ligation, he died.

Postmortem Examination of Cochlear.—Right Side: Bony labyrinthine capsule normal. "There is new connective tissue and bone formation in the scala vestibuli, which is practically obliterated. Marked new bone formation is present in scala tympani in the basal coil, but in the upper coils the scala is practically normal. Cochlear canal is greatly dilated in all coils. Corti's organ is merely a low layer of cells."

Left Side: Very similar changes. Scala vestibuli filled by new connective tissue. Corti's organ is "a low mound of undifferentiated cells." Cochlear nerve in the modiolus is almost completely atrophied.

Fraser says of this case that it "is undoubtedly one of acquired deaf-mutism resulting from (intro-uterine) suppurative labyrinthitis." The grounds upon which this belief is based, though clearly stated and interesting, do not appeal to the author as altogether convincing. In support of the opposite view,—*i.e.*, that this is a case of purely congenital origin,—may be cited the following facts: (1) even a unilateral suppurative labyrinthitis is a comparatively unusual lesion, a suppurative invasion of both labyrinths being exceedingly rare: (2) an intrauterine suppurative labyrinthitis would be exceedingly difficult to determine definitely except in the case of a still-born child; (3) in the light of the great diversity of lesions established by authenticated postmortem examinations of deaf-mutes, there is hardly a conceivable abnormality which may not occur as a congenital variation (see foregoing paragraph on recorded autopsy findings); (4) in the right cochlea there were marked hyperplastic changes in the basal coil of the scala tympani; but "in the upper coils the scala is practically normal," which one would hardly expect if resulting from a diffuse suppurative labyrinthitis; (5) the similarity of postmortem findings in the two labyrinths would seem to suggest in this case a developmental defect or error rather the result of a purulent invasion. These facts show at least another viewpoint, and may serve to stimulate the reader to examine for himself Mr. Fraser's views as to the nature of this interesting case, and further to persue his scholarly discussion of the whole broad subject of deaf-mutism.

The pathologic changes in cases of acquired deaf-mutism show such lesions to consist chiefly of two main varieties: (a) Partial or complete destruction of the membranous labyrinth due to an infective process invading the labyrinth; and (b) degenerative change (atrophy) of the eighth nerves, secondary to meningitis.

In view of the nature of the lesions above outlined, I cannot share Dr. J. Kerr Love's optimistic view as to what may be learned of the pathology of deaf-mutism from physical examination of the ears of living patients.

Treatment.—Deaf-mutism in the vast majority of cases is not amenable to treatment. The only possible exceptions to this rule are found in cases in which islands of hearing persist which by local treatment may be improved to the point of bringing the patient within the category of those who may be taught in part at least through the ear. Ordinarily, as soon as deaf-mutism is positively diagnosticated, the child, having arrived at a suitable age, should be entrusted to trained instructors of the deaf. In the case of the children of the poor, the institution or school for the deaf and dumb at present offers the best opportunity. With the wealthy or well-to-do, the education of the child by specially-trained teachers, away from constant association with other deaf-mutes, probably gives the little unfortunate a better preparation for a useful and contented life. The statement of Dr. Hudson-Makuen, that the development of a hearing mother into a trained teacher, acting under and supplementing the work of professional teachers of the deaf, provides the ideal environment for a deaf-mute child, seems altogether rational. Where the mother lacks the inclination or will-power to play this rôle of love, it may be that some other individual may be inspired by the human need to undertake the task.

CHAPTER XXVII.

NASOPHARYNGEAL ADENOIDS.

The so-called nasopharyngeal tonsil is a physiological structure. Only when it becomes unduly enlarged can its presence be looked upon as an abnormal or pathological condition.

Normally it cannot be appreciated by inspection by reflected light nor does it produce any appreciable symptoms. When, however, through prolonged engorgement of its vessels and consequent increase of its connective tissue, it becomes sufficiently enlarged to produce the smallest degree of obstruction to nasal respiration, it must be regarded as an abnormal condition involving many dangers to the patient's future development and physical well-being.

While the presence of postnasal adenoids is a condition especially characteristic of childhood,—increasing in size during the earlier years, remaining perhaps stationary from the eighth or the tenth year to puberty, and then gradually receding,—this is by no means an invariable sequence of events, the presence of large post-nasal growths in adult patients being not very uncommon. It is during childhood, however, that the most serious and permanently injurious effects of post-nasal obstruction are produced, and it is in childhood that their neglect by parents or family physician is most dearly paid for by the little patient, or, if payment is postponed, it hangs over him to be met, with compound interest, as he grows older.

Pharyngeal adenoids in children are almost invariably accompanied by some degree of hypertrophy of the faucial tonsils; and, *per contra*, noticeable enlargement of the faucial tonsils is one of the most reliable indications of hypertrophy of the lymphoid tissue in the nasopharynx.

Diagnosis.—Cases in which the pharyngeal growth is of such size and so situated as to produce very great mechanical obstruction to nasal respiration may present changes of facial expression so pronounced, characteristic, and familiar as hardly to require description. When the child habitually breathes through the mouth, wears a dull, listless, and somewhat vacant expression of countenance, presents symptoms of nasal catarrh, gives evidence of being either mentally behind his fellows or is hard-of-hearing,—with such a picture the physician's mind at once reverts to a nasopharyngeal growth as the most probable of underlying causes. These are not the cases involving most serious risks to the child, for in the prominence and multiplicity of symptoms there is ample assurance of prompt and adequate treatment.

Unfortunately, in only a very small percentage of cases are such pronounced and unmistakable physical signs present. In the great majority of cases the growth causes only partial obstruction to nasal respiration

and is accompanied by but few symptoms, and these not always pathognomonic.

I shall not attempt to give an extended list of all the minor symptoms and physical signs of a nasopharyngeal growth, but shall content myself with stating briefly what are to me the reliable and all-sufficient indications of its presence.

(1) *Inferential.*—In the first place, a child under eight or ten years of age, with intact drum membranes, rarely becomes hard-of-hearing except as a result of some lesion mechanically interfering with nasal respiration. Pharyngeal adenoids represent by far the commonest obstructive lesion in children. Barring therefore labyrinth deafness, either congenital or acquired, impaired hearing in children is a very strong inferential sign of nasopharyngeal obstruction.

(2) *Retraction of Both Drum Membranes.*—Physical examination of the ears of children suffering from adenoids will show in the vast majority of cases pronounced retraction of both drum-heads. The degree of retraction due to this cause in children is such as is rarely seen in adult life,—never except in the case of extreme atrophy of the membranes. This is one of the most constant and reliable physical signs of postnasal obstruction in children. It is present in many cases in which other indications are inconspicuous or absent.

(3) *Hypertrophy of the Faucial Tonsils and Presence of Granulations upon the Posterior Pharyngeal Wall.*—Personally I have rarely, if ever, seen any considerable degree of hypertrophy of the faucial tonsils in a young child which was not accompanied by an appreciable grade of lymphoid hypertrophy in the nasopharynx. The presence of granulations along the posterior pharyngeal wall is pathognomonic of postnasal adenoids. The combination of these two conditions in many cases renders digital examination quite superfluous,—*i.e.*, so far as determining the presence of adenoids is concerned.

(4) *Posterior Rhinoscopy.*—The inspection of the nasopharynx by means of the small postnasal mirror (pages 72–73) is impracticable in the case of many children. In the case of children old enough or tractable enough to permit it, the growth will usually be seen as an obstructing mass in the median line, or, if of the smooth variety, presenting an apparently unbroken surface with the posterosuperior pharyngeal wall, its presence may be recognized by the fact that the upper portion of the posterior border of the nasal septum is shut off from view.

By digital palpation the growth is recognized as a more or less soft mass which may be felt between the finger and posterior border of the nasal septum, and may also in some cases be recognized in the fossæ of Rosenmüller.

Treatment.—The treatment of adenoids is their surgical removal. There is no question that rather harsh methods resulting in injury either to the pharyngeal wall or to the pharyngeal orifices of the Eustachian canals, or both, have in the past been more or less in vogue. The heavy

use of a curette can remove not only lymphoid tissue but also muscle tissue, giving rise to cicatricial contractures permanently disturbing the function both of the pharyngeal mucosa and of the Eustachian canals.

These exaggerated methods have happily of late undergone modifications under a better appreciation both of the damage which may be wrought and of what the operation is intended to accomplish.

Before ending this brief discussion, I wish to record my personal view in regard to a somewhat mooted point,—viz., the question of removing the tonsils and adenoids in patients suffering from acute purulent otitis media. There are many who still regard an acute tympanic lesion as a contra-indication to immediate operation. I am personally inclined to hold the opposite view, and to believe that, when a pharyngeal growth is clearly a causal factor in acute tympanic disease, the removal of the growth and incision of the drum membrane should be done at the same time. This does not mean that in every case of acute otitis media we should search the nasopharynx for evidences of adenoid hypertrophy, but simply that, when a growth in that situation may act as a hindrance to tympanic resolution, the acute stage of a tympanic inflammation offers a favorable time for its removal rather than a contra-indication thereto. In support of this view may be cited the following facts:

(1) The operation of adenectomy will in a small percentage of cases induce an inflammatory reaction within the tympanum. It seems wiser, therefore, to operate during the acute stage of an existing otitis media when the ears can be safeguarded by free incision of the drum membranes, rather than wait and incur the risk of recurrence as a result of a delayed operation.

(2) The free abstraction of blood from the pharynx which always occurs during adenectomy usually relieves tubal congestion and hastens tympanic resolution.

(3) With a pharyngeal growth sufficiently large to perpetuate nasopharyngeal congestion, recovery from acute tympanic disease is apt to be slow, and not in the final outcome complete.

(4) In the experience of every aurist there are certain cases of acute otitis media in which all therapeutic measures fail until the nasopharynx is cleared of adenoid tissue. Delay in such cases means added risks.

(5) Either myringotomy or adenectomy should be done with the patient under a general anæsthetic. Combining the two operations obviates the necessity of repeated anæsthetization.

The Faucial Tonsils.—In the great majority of cases,—almost invariably in my experience,—the faucial tonsils should be removed at the same time as the adenoid growth in the nasopharynx.

CHAPTER XXVIII.

TUMORS OF THE EIGHTH CRANIAL NERVE.

Forward.—Since the present chapter represents an enlargement of the heretofore prescribed scope of text books on otology, a word may be said as to the reason for this departure from the classical arrangement. Perhaps the best excuse for the inclusion of this additional matter is the fact that, from the viewpoint of the needs of practical otologists, it has become a necessity.

Professor Harvey Cushing of the Harvard Medical School gives the following data based upon the statistics of cases drawn from his own surgical experience: Of 468 tumor cases, in which the lesion has been verified either by operation or post mortem examination, 334 were supratentorial and 134 subtentorial. Of the latter fifty-six were extracerebellar, and of these extracerebellar tumors, thirty "arose from the nervus acousticus." Roughly speaking, therefore, 23 per cent. of all subtentorial, and 6 per cent. of all intracranial tumors of this series were tumors of the 8th nerve. These figures were taken from his monograph published in 1917. In a comparatively short paper published three years later, these percentages as a result of cases subsequently verified were increased to 24.5 per cent. and 7. 3 per cent. respectively.

In his more recent paper, after an allusion to the diagnostic failures of neurologists as compared with those of otologists, Dr. Cushing says: "But our mistakes, as will be seen, are more often from incorrect localization, whereas yours (*i.e.*, of otologists) are more often *due to the failure to recognize the presence of a tumor at all.*" This seems hardly quite fair since at the stage at which these patients are first seen by the aurist, and often for a considerable period therafter, the clinical picture is as a rule not sufficiently developed to bring definite and positive diagnosis within the range of possibility.

Dr. Cushing is on surer ground when, after recounting in detail a number of case histories of patients suffering from acoustic neuromata, upon whom various ill-advised operations upon nasal septum, turbinates, frontal and ethmoid sinuses, etc., had been performed, he says: "*Certainly during this period we can at least spare the patient from meddlesome surgery in the futile effort to improve the hearing.*" Probably in the aggregate of oto-laryngological practice the arraignment herein implied is not wholly unjustified.

The author personally believes that, with the knowledge now at our disposal, the otologist, even though an early positive diagnosis may be, and usually is, impossible, should at least be able to recognize certain features as sufficiently characteristic of this lesion to prompt him to hold such a diagnosis tentatively in mind; to cause him to watch for later components

of the gradually developing symptom-complex, and possibly therefore to modify his management or treatment of such a case..

If this view is justifiable, we reach at once the obvious corolary that the acquisition of a clearer and more universal knowledge of this lesion is not only desirable, but is actually incumbent upon all practical otologists.

Etiology.—Of the causes of these peculiar neoplasms but little need be said, since so little is actually known.

Trauma is by most observers given as the exciting cause in certain cases for the reason that the history of an injury to the head has been elicited in a certain proportion of cases. In many cases the trauma has antedated by many years the incidence of initial symptoms, and the causal relation of the accident to the nerve lesion has by no means been proven. In Cushing's first thirty cases, there was a history of injuries, blows, falls, etc., in six, and in most of these cases the occipital region was the site of the injury.

Age seems to exert no definite influence, beyond the fact that it may be said, in so far as recognizable symptoms are present, to be a lesion of adult life,—*i.e.*, that it occurs in people of adult ages ranging from 20 to 50 or 55 years, and is comparatively rare in old age.

Sex seems to play no part in susceptibility to the disease, since the statistics based on series of cases collected from the literature by different observers yield opposing or contradictory results.

Cushing quotes Sternberg, Verocay, Henschen and others, as holding the view, to which he also inclines, that the lesion may be regarded as of biologic, or embryonic, origin; or in other words, that it may represent what is spoken of as an "embryonic rest." According to this view, the tumor results from the persistence of embryonal connective tissue, within the sheath of the acoustic nerve trunk.

The tumor may involve primarily any part of the nerve trunk, the commoner site being the peripheral portion,—*i.e.*, in or near the internal auditory meatus.

Terminology.—*Author's Note*.—There is apparently considerable confusion, or at least a confusing multiplicity, of names which have been applied to tumors of the 8th nerve. Among them we find fibrosarcoma, glioma, fibroma, neuroma and endothelioma as the commoner designations, with fibroglioma, neurofibroma and gliosarcoma as the next in frequency. Sarcoma, endothelial sarcoma and neuroma fasciculare are also mentioned by Cushing as having been applied to neoplasms presumably originating in the 8th nerve. The confusion, if such it may be called, arising from this extensive nomenclature would not be insurmountable, if it could be assumed that the various designations employed were indicative, or truly descriptive, of actual structural differences in separate and distinct types of tumor. A comparison of terms with the descriptions and microphotographic reproductions of neoplastic tissue accompanying different case reports, leads one to infer, however, that the various designations refer in many in-

stances not so much to actual structural differences, as to differences of interpretation by different observers.

Pathology.—In their most typical form, these tumors are described as firm and cartilaginous in texture, rather dry and but sparsely supplied with blood vessels, which are almost wholly of the venous or capillary type.

Under the microscope the most characteristic appearances are of interlacing fibrous bands, separated by areas of loose reticular tissue. These typical structures are usually well demarcated, the one from the other.

The fibrous bands consist largely of elongated cells with oval nuclei, which tend strongly to an arrangement in more or less parallel lines. The fibrous elements have also the characteristic feature of arranging themselves in relatively straight or waving parallel lines ("palisade" arrangement), or in whorls. According to Councilman, these bands are "in part composed of fine wavy fibrils; in part the fibrils are fused together, the tissue appearing homogeneous."

Separating the fibrous portions, or bands, and usually clearly differentiated or demarcated therefrom, are areas of greater or less extent of loose reticular tissue. The reticular areas are rather scantily supplied with cells of round or irregular contour. The structural resemblance of this reticular tissue to neuroglea and the occasional detection therein of glia-like fibrils—*i.e.*, fibrils closely resembling the fine processes of glia cells proper—give to those acoustic tumors in which the reticular tissue preponderates their chief point of resemblance to typical gliomata.

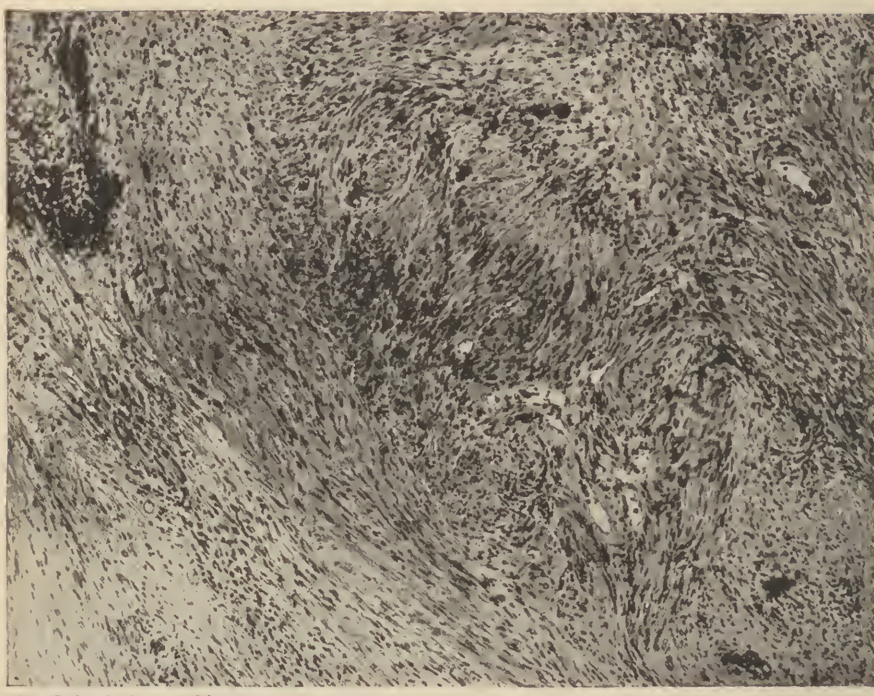
The blood vessels, mainly of venous or capillary type, are particularly subject to hyaline degeneration. The hyaline deposition may occur both within and without the vessels, its most typical site being within the lumen, which finally is occluded. This change is said (Councilman) to be very commonly associated with small areas of necrosis.

There is apparently a considerable tendency to cyst formation within the tumor body, and superficial or peripheral arachnoid cysts have been noted in many cases.

The accompanying illustrations (Figs. 334, 335, 336), show better than any amount of descriptive text, the typical histologic or structural characteristics of these tumors.

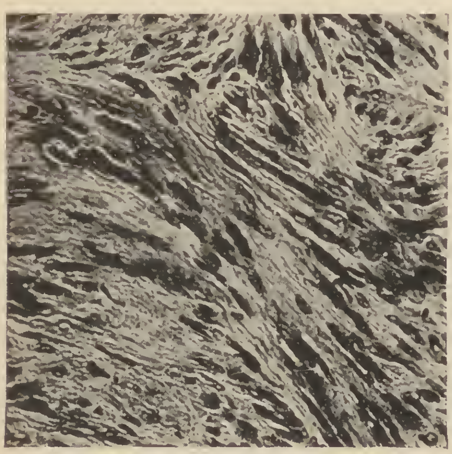
It is clear that great variations may occur in different tumors, otherwise essentially the same in type, in the relative amount of fibrous and reticular tissue present; and also, that a relative preponderance of either element might determine the designation applied to a particular growth. It is also conceivable that the pathologist, receiving his impression from a particular section or field showing chiefly fibrous tissue, might interpret a tumor as a "fibroma," whereas his impression from another specimen or field of the same neoplasm, showing chiefly extensive areas of reticular tissue, might lead to a diagnosis of "glioma."

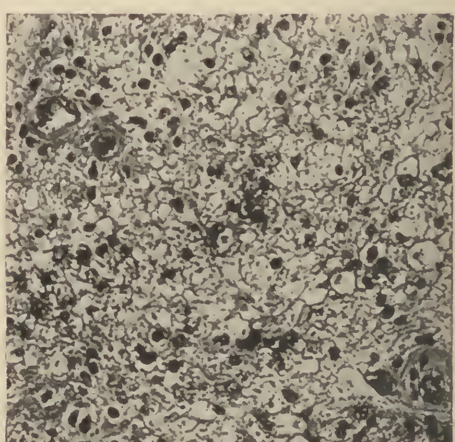
As with variations in ratio between the two types of tissue chiefly composing these growths, so also are there occasional variations in the disposition of cells and fibres composing these tissues. Thus Cushing states that



(From Cushing's "Tumors of the Nervus Acousticus.")

Fig. 334.—Area of tumor showing (× 80) the characteristic architecture of the fibrous areas (hæmatoxylin eosin).





(From Cushing's "Tumors of the Nervus Acousticus.")

Fig. 335. Fig. 336.

Figs. 335, 336.—Characteristic architecture of fibrous and reticular areas.

in certain tumors, otherwise of the typical acoustic neuroma type, the cells composing the fibrous areas or bands, are in certain regions massed so closely and in such numbers as to justify a pathological diagnosis of fibrosarcoma. Again, Councilman referring to the fibrous element of acoustic tumors says: "these bands often form whorls converging to a single point, as in spindle-cell sarcomas." The interesting and fortunate fact in regard to these observations, is that these appearances do not appear in 8th nerve tumors to have the same significance, as would be attached to them in tumors in other parts of the body. That is to say, they convey no special indication of malignancy.

The difficulty in arriving at a correct pathologic interpretation and the importance or necessity of careful examination of more than one or two sections of the same growth are clearly indicated in the pathologic notes accompanying Dr. Cushing's case-histories. These in many instances, give a first interpretation, and a later corrected "diagnosis," based on subsequent examinations. Thus in cases XVI, XIX and XX, the first interpretation of "glioma" was later changed to typical "acoustic neuroma." Cases X and XI which were first called "fibrosarcomata" were found later to be merely structural variations of the usual "acoustic neuroma" type. In a number of Dr. Cushing's cases, the first laboratory interpretation was that of "glioma." In only one case (case XXVI) was the diagnosis of "undoubted glioma" allowed to stand, the basis therefor being as follows: "the entire basic substance is reticular, containing definite neuroglea fibrillæ. The predominant neucleus is small and round with no circumscribed cytoplasm . . . no suggestion of fibrous tissue."

To Epitomize.—Two facts seem to be established with regard to tumors of the 8th nerve: (1) that the most characteristic type of acoustic tumor presents under the microscope, certain structural features which do not occur in tumors originating in other parts of the body; and (2) that in certain cases, tumors of the 8th nerve present in certain parts a structural resemblance to sarcoma, suggesting a malignancy which, however, does not appear to be borne out clinically. In other words, such cases do not appear to follow a particularly malignant course clinically, although all acoustic tumors, probably from their pressure effects upon contiguous structures in the so-called cerebello-pontile angle, tend regularly toward a fatal ending.

So much for structural features of these tumors. Of much greater importance to otologists,—*i.e.*, in their relations to the clinical picture—are the gross changes wrought by pressure or displacement in neighboring structures.

It is clear from case histories and post-mortem reports from many sources, that the vast majority of 8th nerve tumors originate from the peripheral end of the nerve trunk, *i.e.*, in or near the internal auditory meatus. Henschen was so much impressed by the almost invariable presence within the meatus of a part of the tumor growth—*e.g.*, as established by his own observations and the post-mortem reports of other observers—as to incline him to the belief that all acoustic tumors originate within the

meatus. Cushing states that "Alagna's case is one of a very few in which it is specifically stated that there was not the slightest projection of the tumor into the porus." He reviews the literature as to the almost invariable presence within the meatus of a part of the growth, and cites cases (Virchow's, Balance's, Askanazy-Funkelstein's, Panse's) in which some dilatation of the meatus from pressure absorption had occurred. His own conclusions are that, while the vast majority of acoustic tumors have their origin in the peripheral end of the nerve trunk, and while practically all such growths in their later stages send projections into the internal auditory meatus, that space cannot be regarded as their invariable point of origin.

As might be expected, acoustic tumors are approximately oval in contour in the early stages of their development, though naturally, with their gradual increase in bulk, they must conform somewhat to the unyielding angular space between the tentorium and postero-internal surface of the petrous bone, while internally and backward they present a nodular surface which bulges against the comparatively unresisting surfaces of cerebellum and pons.

Influence of Tumor Upon Other Nerves.—All the cranial nerves, from the 5th to 12th, are in close proximity to the space usually occupied by a fairly large acoustic tumor. While all or any of these may be subjected to pressure, the facial and trifacial are the two nerves which commonly suffer the greatest distortion. The facial trunk being always in relation to the upper end, or pole, of the tumor may be elongated to an extent three times its normal length or more. The trigeminus, which also is pressed upon by the upper end of the growth, may be similarly stretched. Cushing states that it is a matter of wonder, the degree to which these nerves may be elongated, flattened and attenuated without appreciable disturbance or loss of function. This is the more surprising in the case of the 7th nerve since in addition to stretching and displacement, it must also be subjected to an unusual degree of pressure within the internal auditory meatus. Probably this rather remarkable retention of function in a motor nerve is to be explained by the very gradual development of pressure, the nerve fibres having time to accommodate themselves to the gradual changes in structural arrangement or relationship. Unquestionably, any like degree of pressure or distortion, suddenly applied, would result in immediate, and complete, though not necessarily permanent, paralysis of the supplied facial muscles. The abducens, though from its position apparently less subject to pressure than any of the cranial nerves mentioned, is never-the-less paralyzed occasionally in cases of 8th nerve tumor. At their points of emergence from the medulla the glossopharyngeal, pneumogastric and spinal-accessory nerves are in fairly close relation to the 8th nerve, though diverging from and passing below the latter in their course toward their point of exit from the skull through the foramen lacerum posterius. They are, therefore, pressed upon by the lower end of an acoustic tumor and may undergo great elongation and distortion.

Influence of Tumor Upon Pons, Cerebellum and Medulla.—Naturally a tumor so situated as to exert direct pressure inward upon the pons, inward and downward upon the medulla and inward and backward upon the cerebellum, may cause great displacement and distortion of these structures. Cushing states that in cases in which a wide decompression operation for the relief of tension has been performed, the acoustic tumor in certain instances reaches a size or length rarely or never seen where the skull remains intact; and the fact of this retardation of tumor growth as a result of its confined space, gives us a clear conception of the enormous pressure to which the cerebellar, pontile and medullary structures are themselves subjected. It is not surprising, therefore, that in the later stages of such a growth, the adjacent half of the pons may be compressed to half its normal size, the cerebellar hemisphere deeply impressed and even the medulla indented and displaced. Add to the pressure of increasing tumor bulk the influence of distended ventricles and the displacement of the whole brain stem toward the opposite side, the compression, or herniation, of cerebellar tissue downward toward or even into the foramen magnum and the pressure absorption of the petrous bone and skull plates are not incomprehensible.

Influence of Tumor Upon Intra-cranial Pressure.—No clear or comprehensive picture of the gradually developing symptom complex is possible without an intelligent consideration of the enormous increase of intra-cranial pressure to which this lesion may give rise. This increase is due not only to the direct influence of the increasing tumor bulk but also to the disturbance of free circulation of cerebral fluids with consequent ventricular distension. It must be borne in mind that we are considering a subdural growth lying, as Cushing expressed it, "within an arachnoid capsule." He calls attention to the fact that while this may be difficult of demonstration post mortem when the membranes have been disrupted during removal of the brain from the skull, it is usually quite obvious during operation "when the arachnoid spaces are distended with fluid."

Barany has described the train of symptoms due to distension of what he calls the cisterna pontis lateralis. This space lies in the so-called cerebello-pontile angle,—or, to be more exact, is in close relation to the internal auditory meatus, and that portion of the postero-internal surface of the petrous bone immediately surrounding it. Within the lateral cistern are found the glossopharyngeal and vagus nerves, the lateral choroid plexus, and the facial, acoustic, and trigeminal nerves, all of which structures may suffer compression or distortion by an acoustic tumor. Barany assumes that this space, which has communications with the other ventricular reservoirs of the brain, may become distended through inflammatory adhesions between pia and arachnoid with consequent disturbance of the circulation of the cerebral fluids; and he describes a group of symptoms—nystagmus, vertigo, ataxia, loss of normal pointing reactions, etc.—all of which may form part of the characteristic symptoms-complex of a tumor of the 8th nerve. If such disturbances are possible as the result of a simple inflammatory process, one should have little difficulty in conceiving the

enormous and generalized pressure changes through ventricular distension, which may be produced by a tumor which by direct or indirect pressure may close or obstruct the foramena of Luschka and Monro, and possibly also the aqueduct of Silvius. This degression is made here solely for the light it may throw upon an important factor in the symptomatology of this lesion.

Symptoms.—Cushing lays great stress upon the importance of the "chronology," or the order in which the symptoms of an acoustic tumor appear; and while this important factor in the diagnosis has, so far as the writer knows, not been duely emphasized by other writers, the observation accords so logically with the histologic and gross pathology of such a lesion as to carry immediate conviction of its accuracy. In fact it would almost seem that, without any knowledge of symptoms, one might deduce that a slow-growing tumor originating in the trunk of a nerve of special sense would proclaim its presence: (1) by functional disorders of the organ supplied; (2) by symptoms of intracranial discomfort; (3) by symptoms induced by pressure upon, or displacement of, adjacent structures,—*e.g.*, nerves or organs; (4) by symptoms referable to increase of intracranial pressure, *e.g.*, pressure upon brain centres, more distally placed nerves, etc.

Deafness.—Almost of necessity the first appreciable symptoms of an 8th nerve tumor are those referable to the nerve itself, *i.e.*, impaired hearing, homolateral and at first not sufficiently pronounced to alarm the patient. While qualitative analyses of the auditory defect seem to be entirely lacking, it is inconceivable that the type of impairment can be other than that known to otologists as "nerve deafness." In support of this deduction is the recurring statement in Cushing's case-notes that the patient's attention was first directed to a loss of hearing by *difficulty in using the telephone*, which is distinctly not the case with the commoner type,—*i.e.*, of obstructive deafness.

Tinnitus aurium is also one of the usual symptoms of this early stage. Probably in many cases, the subjective noises represent the first indication of aural disorder.

The deafness in acoustic tumors has the following characteristic features: it is of nerve or labyrinthine type; it is uninfluenced by local treatment; it is progressive and probably very rapidly progressive, and tends regularly, though with some exceptions, to complete loss of function.

Vestibular Disturbances.—That a lesion originating in the nerve trunk, and believed by some (Henschen) to involve primarily the vestibular branch, should produce no onset-symptoms of vestibular upset or disturbance, would seem at first almost incredible. Case reports are so contradictory as to this phase of the syndrome, however, that it will be reserved for a brief later consideration. Undoubtedly some evidence of vestibular involvement must be present at the onset in the great majority of cases.

Note.—It may be said with approximate certainty, that deafness, tinnitus and possibly vertigo, are the initial symptoms in practically all cases of acoustic tumor, although their importance may have impressed

the patient so little, that he may have completely forgotten the date of their onset when the next and more alarming components of the syndrome make their appearance. It is this long waiting period—possibly measured by years—between the unspectacular onset and the further development of symptoms, which makes an early diagnosis so exceedingly difficult a problem.

Pain.—Next in order of sequence, and certainly representing a stage of tumor development very considerably later than that of the initial deafness, are certain subjective sensations of distress, discomfort or actual pain in the head. These may take the form of inconstant and rather indefinite pain or may reach a degree of considerable severity. They are usually homolateral and occipital, or may be frontal. They may alternate between the occipital and frontal regions, but more often have their point of greatest severity and persistency in the subtentorial region with radiating or shooting pains in the direction of the frontal region. They are subject to periods of relief or amelioration; they are usually worse at night, or may afflict the patient only at night, subsiding wholly in the morning or as the day advances.

Naturally with a subjective symptom of such variable and, in some cases, intermittent type, its diagnostic value, unless supported by other evidences of intracranial disease, may be slight. Usually, however, other characteristic phenomena may be detected. With the progress of the lesion the head pains tend to become more definitely localized and of greater severity; and with their gradual accentuation, the homolateral occipital and neck muscles in some cases become markedly sensitive or tender,—so that the patient, to relieve tension, holds the head tilted somewhat to the affected side.

Cerebellar Symptoms.—As one would expect, symptoms induced by compression or distortion of cerebellum, pons Varolii or medulla are of later development than those referable to the pathologic changes in the 8th nerve itself. From the author's analysis of case reports he is inclined to believe that they may appear at any period from 2 to 10 years after a correctly noted initial deafness. It must be borne in mind, however, that a unilateral deafness often excites little notice so that in certain cases the graver symptoms may appear to follow quickly upon the first appreciation of auditory loss.

The chief cerebellar symptoms are: nystagmus; incoördination ataxia and diadokokinesis; static and dynamic ataxia.

The *nystagmus* apparently varies considerably in different cases. From the descriptions, one receives the impression that in a majority of cases the direction is toward the side of the acoustic tumor, and that it becomes more pronounced when the eyes are rotated in that direction.

Homolateral incoördination ataxia of the arm and leg muscles and homolateral *diadokokinesis* are almost always present. They are probably to be regarded as different manifestations of the same disturbance rather than separate symptoms. Probably homolateral paresis of the affected

muscles is usually present, since this is a common concomitant of the incoördination ataxia and diadokokinesis of all cerebellar lesions.

Static and Dynamic Ataxia.—The gait is markedly unsteady. The patient may be able in walking to pursue a straight course, but does so with effort and with feet well apart; or he may be quite unable to stand or walk, the falling direction or tendency being usually toward the side corresponding to the lesion.

The tendency of the patient to fall toward the side of the lesion is probably to be accounted for in one of two ways: (1) the homolateral incoördination, semiparesis and diadokokinesis would naturally tend, through the resulting lack of support and resistence, to throw his body in that direction; and (2) in addition to the above or, it may be, quite independently, there may be an abnormal impulse, resulting perhaps from pressure upon, or injury to, some part of the complex mechanism of orientation,—basal centre, association tract or possibly a so-called direction centre in the cerebellar cortex (Barany), which impels the body in the given direction.

Naturally with objective disturbances of equilibrium so pronounced, subjective vertigo of a distressing type is likely to be present.

The above so-called cerebellar symptoms occur regularly at some stage of an acoustic tumor in the great majority of cases. and may be generally regarded as signifying a growth which has already attained a very considerable size.

Before leaving this brief outline of cerebellar phenomena, a word may be said, from the otologist's viewpoint as to the differential value and significance of nystagmus and ataxia as seen or reported in the course of this lesion.

Nystagmus.—From the detailed reports of Dr. Cushing's first series of thirty cases—unusually complete in most respects—we learn but little as to the nystagmus beyond the meagre fact that it is almost invariably present, its absence being noted in one case only (case vii). Such statements as "nystagmus, coarser excursions to right" (case ii), "nystagmus, slower to left" (case iii), "nystagmus, coarser to left" (case iv), etc., recur frequently as the only qualifying term. In only two of the thirty cases (cases XX and XXI) is the direction of the quick component of the nystagmus given. From the otologist's viewpoint, the mention of a nystagmus in connection with either a labyrinthine or a brain lesion without definite statement or indication of the direction of its quick component, is to deprive it of its chief diagnostic significance. Dr. Cushing's histories are and should be of such interest to otologists, and the character of the nystagmus is from the otological viewpoint so essential to a correct interpretation of the accompanying phenomena of vertigo and ataxia, that it is to be regretted that he has not been sufficiently interested in Barany's work on the labyrinth and his later work on cerebellar localization to have adopted terms which would convey a distinct and definite meaning to otologists and neurologists alike. In other words, if this lesion is to come under the

joint study of neurologists and otologists, a code of terms intelligible to both is clearly essential.

More than once in his treatise on acoustic tumors (pp. 155, 156 and 161), Dr. Cushing speaks of the question of a vestibular or cerebellar origin of the characteristic nystagmus and ataxia of the later stages of these neoplasms as one often difficult of solution. Yet from the otological viewpoint it would seem that this question should not present great difficulties, and for the following reasons: (1) in most of these cases the evidence of early paralysis of the vestibular nerve seems complete, and active nystagmus and ataxia from a functionally inactive vestibular nerve is almost invariably of short duration. In other words, in the severest types of vestibular lesions, *e.g.*, suppurative labyrinthitis, the nystagmus and accompanying disturbances of equilibrium usually subside quite rapidly. (2) If, as Dr. Cushing believes, the involvement of the vestibular nerve trunk occurs in many cases without any onset-symptoms of vestibular disturbance, it is inconceivable, in the light of what is actually known of vestibular physiology and reactions, that such phenomena could occur as a late manifestation of such a lesion. (3) In any given case, spontaneous nystagmus and static ataxia both being present, a study of the ataxia, *i.e.*, relation of the falling direction or tendency to the direction of the quick nystagmic component, to changes in the position of the head, etc., should suffice either to establish or eliminate the diseased vestibular mechanism (nerve trunk or labyrinth) as responsible for the symptoms in question.

Symptoms Referable to Disturbance of Adjacent Nerves.—These in the order of their diagnostic importance are the 5th, 7th, 6th and then the group of nerves (9th, 10th and 11th), leaving the skull through the foramen lacerum posterius.

The 5th, or trigeminal, nerve is given precedence over the 7th, for the reason that the symptoms referable thereto, on account of their subjective character, are more surely brought to the physician's attention and therefore less likely to escape notice.

Fifth Nerve.—The trigeminal symptoms are chiefly sensory, consisting in their most characteristic form of a paræsthesia,—usually a sensation of numbness, or in some cases of numbness and tingling combined—in the entire trigeminal area of the anterior and antero-lateral surfaces of the head and face. Closely associated with these sensory pervertions in the skin is a loss or diminution of the corneal reflex, which is perhaps the commonest manifestation of trigeminal involvement in cases of acoustic tumor. Cushing instances the fact that in four cases of his series areflexa cornealis supplied the only evidence that the 5th nerve was involved, and in one case it was the only indication that any cranial nerve, other than the auditory nerve itself, was in any way affected.

It will be seen from the above that the typical symptoms of trigeminal disturbance present the characteristics of a nerve paralysis or diminution of function. The opposite phenomena of nerve irritation, or pain, are comparatively infrequent. Slight neuralgic symptoms have been

noted in some cases. But severe neuralgia—*i.e.*, neuralgia of characteristic trigeminal type—is said to be uncommon.

While all of the sensory phenomena of 5th nerve involvement are typically and logically homolateral, several instances have been noted in which they occurred bilaterally, a condition presumably due either to extensive contralateral displacement of the brain stem, or quite possibly in some cases to the pressure of the coincident hydrocephalus.

Seventh Nerve.—That the facial nerve, subject as it usually is not only to stretching and attenuation, but also to compression within the internal auditory meatus (see pathological notes), should not induce earlier and more pronounced signs of paralysis, affords a graphic illustration of the extensive changes which may occur in a motor nerve—provided these changes are gradual in their development—without complete ablation of function.

While some facial paresis, shown by slight loss of motor activity and diminished clearness in the facial lines, is almost always present, it may be so slight as to pass unnoticed unless looked for, and complete facial paralysis is said to be exceedingly rare.

More conspicuous, when they are present, are facial nerve disturbances of the opposite or irritative type. Facial twitching, or spasms, have been recorded by several historians of acoustic tumor cases. As compared with the paræsthetic type of phenomena, however, they are extremely rare.

Sixth Nerve.—While the abducens is a comparatively deep-seated nerve, passing almost directly forward from its superficial origin to its distribution to the external rectus occuli, it is to some extent involved in a very considerable proportion of acoustic tumor cases. That the involvement of this nerve is said in most cases to be indicated only by occasional or intermittent diplopia, and that it has occurred in a number of cases bilaterally, seem to the writer to indicate that abducens phenomena may occur chiefly as a manifestation of changing conditions of ventricular pressure,—possibly of intermittent distension of the cisternæ pontis lateralæ.

Ninth, Tenth, Eleventh and Twelfth Nerves.—While all of these nerves undergo almost inevitably a considerable degree of elongation or stretching through displacement by the lower part of a fairly large acoustic tumor, it is probable that complete paralysis, or loss of function, is quite unusual. Even did actual paralysis occur, the resulting symptoms might be difficult to differentiate definitely with regard to their actual causes. Thus with the subjective disturbances of taste, not only the glosso-pharyngeal but also the facial, through its chorda tympani branch, would have to be considered; while marked pulse and respiratory changes might be due either to disturbance of the pneumogastric or to pressure changes affecting medulla. Again, the speech defects which are so commonly present among the symptoms of the later stage, may be due in part to involvement of the motor fibres of the glossopharyngeal, of the inferior laryngeal branch of the vagus, and possibly of certain motor fibres of the spinal accessory supplied to the larynx. To which of these nerves the difficulty in swallowing is

chiefly to be ascribed is probably a matter largely of conjecture. For diagnostic purposes, therefore, it seems better to describe all these symptoms as belonging to a group probably or possibly having a composite origin. They may be mentioned in the following somewhat arbitrary order: *difficulties in swallowing; speech defects*—usually slow, labored articulation; *incontrollable and frequent yawning; occasional paroxysmal attacks of difficult or labored breathing.* Probably all of these symptoms are present at some stage of the development of almost all acoustic neuromata. The dysphagia and disturbances of articulation are said to be prominent and almost invariable phenomena of the later stages of the disease.

A symptom described as occasionally present is the deflection of the protruded tongue toward the side of the lesion. It is ascribed to involvement of the hypoglossal nerve.

General Pressure Symptoms.—Under this head must be mentioned an exceedingly important symptom, which seems to be one of the most unfailing manifestations of the hydrocephalus which usually marks the later stages of an acoustic tumor—viz., *failing vision* with *choked disc,* and possibly beginning optic atrophy. The first indications of failing sight in a patient presenting other characteristic phenomena of an acoustic neoplasm are of the greatest importance, not only for their diagnostic value, but also as indicating a stage of the lesion when surgical intervention must not be unduely delayed if the patient's sight is to be preserved. Dr. Cushing cites at least one instance of permanent blindness in a patient who presented typical symptoms of an acoustic tumor, whose sight might have been saved by timely surgical intervention, but who was subjected to illogical and futile treatment based on a false diagnosis.

Epitome.—The symptoms of an acoustic tumor constitute a gradually developing symptom-complex the usual sequence of which is as follows: (a) homolateral impairment or loss of hearing, probably accompanied by symptoms of vestibular disturbance; (b) head pain or discomfort; if pain, usually occipital, frontal or alternating; tinnitus aurium; (c) cerebellar nystagmus; incoördination ataxia and diadokokinesis; static and dynamic ataxia; (d) paræsthesia—numbness or tingling of trigeminal region of face and head; areflexa cornealis; slight facial paresis; diplopia occasional or intermittent; all symptoms of this group homolateral. (e) Failing vision, with choked disc, leading to optic atrophy; (f) dysphagia; motor speech defects; severe paroxysmal head pains; mental and dispositional changes; attacks of difficult respiration; pulse changes; terminal stupor or coma.

Since the otologist faces a peculiarly difficult problem in regard to these tumors, there are two points in the clinical picture which call for brief discussion,—*i.e.* (a) The question of vestibular symptoms at the onset; and (b) the average duration of the interval between the *acoustic* and the later *cerebellar* or pressure symptoms.

Dr. Cushing's analysis of his own carefully made case-histories discloses so many instances in which no experience of early disturbance of equilibrium or dizziness could be recalled as to raise in his mind a question

as to the frequent occurrence of early vestibular symptoms. One finds it difficult, however, to believe that so gross a lesion of the vestibular nerve could occur without definite symptoms of vestibular disturbance. It is a point for the otologist's observation and study.

Interval Between Initial (Acoustic) Stage and the Later (Cerebellar) Stage.—Definite knowledge on this point—*i.e.*, the average interval period—would be not only interesting but valuable. The author has hazarded the statement that the secondary phenomena might appear at any time from 2 to 10 years after the first aural symptom. Even with so large a spread, however, this statement is largely conjectural. It may be interesting to give a correlation of Dr. Cushing's data on this point. These patients, be it remembered, were first seen by him when the symptom complex was complete. Whenever obtainable, the number of years preceding, or the date, when the initial auditory failure was appreciated is stated on the history chart. These duration periods in twenty-two cases and stated in terms of years are as follows: 3, 9, 5, 2½, 10, 3, 2, 2, 7, 2, 2, 2, 1, 4, 3, 4, 5, 1, 2½, 1, 3, 1; average, 3½ years. This would seem very conclusive but for the fact, known to all aurists, that patients with unilateral deafness are extremely apt to understate the period of its duration. There are two sources of error in such data, *i.e.*, the patient's inability to recognize the impairment until advanced, and errors of memory as to dates.

Every otologist should be able to recognize an acoustic tumor when the syndrome is complete and the chronology of symptoms is known to him; but the problem of reaching even a tentative diagnosis in the earlier stages is a far more difficult one.

Treatment.—In the early stages of an acoustic tumor,—the period of tentative diagnosis,—the real aim of treatment should be, not improvement of hearing, but (a) maintaining the patient's vitality on the best possible plane against the stress and strain of the later period, and (b) careful periodic examinations for detecting any later components of the gradually developing symptom-complex. When the clinical picture has reached the point where a positive diagnosis is possible, the treatment becomes purely a question of surgical experience, judgment and method.

A question which has been discussed, is with regard to a choice between two surgical methods, or two pathways of surgical approach, for removing these tumors: *i.e.*, (1) the wide bilateral occipital exposure advocated by Dr. Cushing; and (2) the more direct route through the tympano-mastoid space and labyrinth, proposed by Dr. Krause. Perhaps the best light upon this question may be in the statement of certain facts relating to the two operations.

In the first place, the mortality following the Krause, or labyrinthine, operation is recorded as 80 per cent. (Fraser); by the occipital exposure and technic of Dr. Cushing it has been reduced to 20 per cent.

The Cushing operation provides a wide decompression area which is in

itself an important factor in the relief of symptoms. It leaves a closed wound with practically no risks of post-operative infection.

The Krause operation involves the maximum risks of facial nerve injury or destruction; does not provide a sufficient exposure for the most careful work upon the tumor; is far inferior to the occipital operation in providing durable relief of tension; and leaves a wound which cannot be closed, can hardly be kept surgically clean, and imposes a maximum risk of meningeal infection.

Dr. Cushing's experience has lead him to the conclusion that in the past the very great mortality—a mortality so large as almost to discourage surgical effort—has been due in part to the very natural desire and effort on the part of surgeons to remove these tumors in *toto*—*i.e.*, with their capsules. The percentage of mortality in his own early experiences led him to substitute for the effort at capsular enucleation, a subcapsular operation. This, he admits, does not effect complete exterpation of the neoplastic tissue, but provides practical relief of symptoms over a considerable period. It is obviously better in any series of 100 cases, to confer an extension of life with comfort upon eighty, rather than a permanent result upon twenty of the series at the expense of the sixty whose lives are sacrificed to the more radical procedure. It must be borne in mind that the operative procedure developed by Dr. Cushing confers an average extension of life estimated at six or seven years and this without prejudice to the chances of relief from a second operation, should this be indicated later.

Henschen estimated the average period of development of acoustic nerve tumors—presumably from the initial recognizable symptom to the fully developed symptom-complex—at 3½ years. This coincides with Cushing's computation that from the initial symptom the patient has before him a period of failing health of approximately 3½ years to the stage where operation becomes a positive necessity. Following a successful operation he may expect a period of 3 to 3½ years of comparative health and comfort, when tumor symptoms reappear, presaging a period of failing health and gathering symptoms of equal duration, when operation again becomes a necessity.

It is estimated that a patient presenting the complete symptom-complex of an acoustic tumor—*i.e.*, having reached the stage of well marked concomitant hydrocephalus—lives on the average less than one year.

Before closing this chapter, the writer wishes to acknowledge his entire indebtedness to the writings of Dr. Cushing for practically all of the facts presented. Those who wish to inform themselves as to the details of the surgical procedure are referred to Dr. Cushing's work on "Tumors of the Nervus Acousticus." No clearer or more beautiful description of a difficult surgical technic is known to the author than will be found in his chapter on the surgical treatment of these growths.

Differential Diagnosis.—There is a rather long list of lesions said to have been confused with tumors of the 8th nerve, but only three which need be mentioned here:—(1) cerebello-pontile tumors not originating in

the 8th nerve; (2) distension of the lateral cistern from inflammatory adhesions between pia and arachnoid; (3) syphilis of the 8th nerve.

Tumors of the cerebello-pontile angle not of 8th nerve origin may naturally give rise to all or any of the symptoms entering into the symptom-complex of an acoustic tumor. The chief differential sign is one of chronology,—*i.e.*, deafness is the initial symptom of 8th nerve tumors whereas it is one of the later manifestations of tumors arising from other structures of this region. In fact, deafness, which is often complete in 8th nerve tumors, may be slight or even unappreciable in other neoplasms—*e.g.*, of cerebellum or pons—in this region.

Distension of the cisterna pontis lateralis may present a clinical picture strongly suggesting an acoustic tumor. It is to be differentiated therefrom by the comparatively rapid development of the complete syndrome, and the later and less pronounced disturbances of the auditory function.

Syphilis of the 8th nerve gives rise to sudden and often complete homolateral deafness with concomitant disturbances of equilibrium,—nystagmus, vertigo, ataxia, loss of vestibular reactions etc. It is to be differentiated from an acoustic tumor by the result of a Wassermann test of the blood and spinal fluid, and by other signs of syphilitic infection.

Naturally an intracerebellar tumor superficially placed in the middle inferior lobe may present many clinical resemblances to an acoustic tumor, but with the important differential point of absent or slight disturbances of auditory function.

In Conclusion: Admitting, as one must, the difficulty or practical impossibility of a positive diagnosis in the phase of initial symptoms, what are the aurist's responsibilities with regard to this lesion in the stage at which he alone is likely to be consulted?

Personally, I should say that, having familiarized himself with the chronology of symptoms, he should consider this lesion—at least tentatively—in all cases of nerve deafness of unexplained origin. The vestibular tests should be made irrespective of the presence or absence of spontaneous symptoms of vestibular disturbance. He should keep the earlier of the secondary phenomena in mind—*e.g.*, of 7th nerve involvement—and should keep the patient under periodic observation for the detection of successive components of the symptom-complex. He should remember that deafness of labyrinthine or 8th nerve type is not favorably influenced either by local treatment of the ear or by nasal surgery. When the clinical picture has reached the stage of adjacent nerve involvement and characteristic phenomena of cerebello-pontile disturbance or pressure, he should recognize the patient's right to the best available neurological opinion and advice. Finally, a diagnosis having been made, he should bear in mind that the lesion is one calling for the best surgical skill and experience, and further that delay may mean the unnecessary and permanent sacrifice of the patient's sight.

APPENDIX.

Aural Disease in Relation to Life Insurance.—As the various aural lesions involve risks which insurance companies have frequently to consider, it may be well to discuss briefly the risks which—from the aurist's point of view—these patients would impose upon the companies insuring them.

I personally believe that the attempt to reduce the solution of this question to a set of dogmatic, unyielding rules would lead in many cases not only to injustice and hardships to the individual, but also to a loss to the insurance companies of a considerable amount of legitimate business. In other words, I believe that each applicant harboring an aural lesion of any form should be examined by a competent and experienced aurist, and his application decided in accordance with a careful analysis of all the data involved in his particular case.

Among conditions which should determine absolutely a rejection of the applicant may be mentioned the following:

I. Malignant disease (epithelioma or sarcoma) of any part of the organ of hearing.

II. Advanced tuberculous lesions of the auricle (lupus hypertrophicus) as pointing to a constitutional tendency unfavorable to longevity.

III. Chronic middle-ear suppuration, with evidences of a diffuse suppurative invasion of the labyrinth, either present or past.

The above conditions should permanently disbar the patient as an applicant for life insurance.

In a second category may be placed:

IV. Chronic middle-ear suppuration with any of the following conditions:

(a) Polypi springing from promontory, region of oval window, or from any part of the inner tympanic wall.

(b) Polypi attached to the inner margin of the osseous meatus (annulus tympanicus) or within the attic, which persistently recur after removal.

(c) Chronic middle-ear suppuration with recurrent exacerbations of acute mastoid inflammation, any one of which may lead to intracranial invasion.

(d) Evidence of cholesteatoma in any part of the tympanomastoid cavity.

(e) Present facial paralysis or a history of past involvement (paralysis) of the facial nerve, not the result of surgical interference.

The above should absolutely exclude the applicant until the disease has been eliminated by surgical intervention. A complete and permanent cure can be determined only by a competent aurist and only after a yeaı has elapsed since complete cessation of all symptoms.

In contradistinction to the above, I believe that there are many cases in which, in spite of a large perforation allowing a certain amount of aural discharge, the character of the lesion is such as to involve little or no risk to life. On competent expert advice, such an individual might well be accepted at an increased premium rate.

The various acute suppurative lesions involving either tympanum or external ear should during their active period absolutely exclude the applicant. They are usually amenable to treatment, and complete recovery usually removes any risk involved in the attack.

In the effort to elucidate this difficult question, many statistics have been collected, a large part of which, while of academic interest, does not supply a basis for practical deductions. For example, the copious statistics from the general autopsy records of large hospitals—*e.g.*, Pitt's analysis of Guy's Hospital Reports, showing one death from aural suppuration to 158 from other causes, and Gruber's analysis of the autopsies in the Vienna General Hospital, showing one death from aural suppuration to 173 from other causes—do not bear very strongly on the risks which these lesions impose upon insurance companies. What we need to know is in just what percentage of all cases does chronic middle-ear suppuration end fatally or shorten the patient's life; and this is a question at present impossible of solution.

The determination, from the records of an institution specialized for the treatment of aural disease, of the proportion of deaths from aural suppuration to the total number of aural lesions treated, does not supply reliable data; for the more serious cases of aural disease and of intracranial lesions resulting therefrom are naturally sent to such an institution, and there is no way of correctly computing the large number of individuals with chronic middle-ear suppuration who are treated in other institutions, by private physicians, or who are content to go without treatment.

Artificial Aids to Hearing.—Among these may be mentioned Toynbee's and other artificial drum membranes, paper disks employed as substitutes therefor, the various ear-trumpets, Rhodes's audiphone, the acousticon, etc.

Toynbee's artificial drum consists of a circular disk of rubber, one side of which is provided with a central loop or eyelet, through which is passed a thread by which it may be withdrawn or controlled. It is used in cases in which a perforation of the drum membrane is supposed to interfere with the efficient transmission of sound-waves. The surface to come in contact with the remnant of drum membrane may be lightly covered with sterile albolene. After it has been applied its position should be shifted, the hearing being tested meanwhile to note the effect upon audition. In some cases functional improvement results; very much oftener no gain can be demonstrated. Obviously its use is contra-indicated in cases of active suppuration,—*i.e.*, in cases in which the perforation provides a necessary pathway for the escape of pus or serum.

A substitute for the above may be improvised by flattening a minute

ball of sterile absorbent cotton, cutting away the edges to give it a circular form, and covering the surface to be applied with sterile vaseline or albolene. This in some cases materially augments the hearing power. More often it does not, and it may collect germs and provide favorable conditions for their growth.

In cases of dry perforations of moderate size, the application of small disks of paper, cut to appropriate size and soaked in alcohol, is sometimes of value, and is infinitely cleaner than either of the foregoing.

Rhodes's audiphone consists essentially of a flexible fan-shaped sheet of hard rubber, one edge of which is placed in contact with the upper incisor teeth. Sound-waves impinging upon it are transmitted through the teeth and cranial bones to the ear. It is a rational effort to utilize the increased bone conduction characteristic of tympanic disease, and will undoubtedly augment the hearing power in many cases.

The various ear-trumpets are mechanisms all of which are designed to provide a larger and more effective receptacle for sound-waves than is the cavity of the concha. When provided with a tube and mouth-piece into which the person he is conversing with directly speaks, its effectiveness is naturally greatly increased. All of the ear-trumpets and tubes are open to the objection, which with many of the hard-of-hearing overbalances their value, that they render the individual more or less conspicuous and do not enable him to take any part in a general conversation.

The aurophone, though of undoubted value to many individuals, seems without value to others suffering apparently from similar lesions. One difficulty with this and similar instruments is that, while the sound of the human voice is intensified, so also are the other multitudinous (but to the patient unappreciable) sounds which constantly surround him,—this resulting in a sort of artificial tinnitus aurium, to which some individuals never become accustomed.

It is regrettable that the application of the various artificial aids to hearing have not yet been placed upon any scientific or practically available basis, enabling the aurist to determine from his aural examination just what instrument will best suit the individual patient. Very largely it is a matter which can be decided only by the patient's personal experiments with the different instruments.

Case Histories.—For the convenience of those wishing to enter upon a systematic clinical study of otology, we append below two history forms, or charts, which have proved most satisfactory in the author's practice.

The shorter of the two (No. 1) was especially designed for bedside histories, and enables the physician to record the essential clinical facts in a case of acute tympanic or mastoid disease with a minimum expenditure of time and effort.

No. 2 is used wholly for recording the results of office examinations, and facilitates the taking of adequate histories in cases of chronic aural disease.

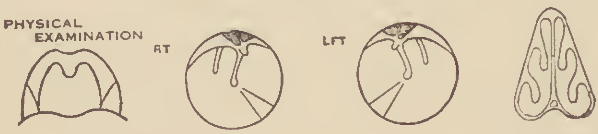
History Chart No. 1. (for bedside histories)

Name Age Date

Address Patient of Dr.

History

Temperations.



Mastoid Tenderness

RIGHT | LEFT

Character of Aural Discharge ; Examined by Dr.

Diagnosis { Right
Left

Treatment

HISTORY CHART No. 2. (for office histories)

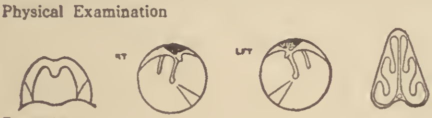
Name ______ Age ______ Occupation ______
Date ______ Address ______ Referred by ______
Symptom for which relief is chiefly sought ______

General History

Diatheses Heredity ______

Aural History

Physical Examination



Remarks

Condition of Eustachian tubes.
RIGHT.
LEFT.
REMARKS

Hearing Tests	RIGHT BEFORE	RIGHT AFTER	LEFT BEFORE	LEFT AFTER
Watch or Acoumeter				
Voice or Whisper				
Lower tone limit				
Upper tone limit				
B. C., fork				
Rinné				

WEBER REMARKS

VESTIBULAR TESTS

Spontaneous nystagmus, Right } .... Left }............
Fistula test }.. ....
Caloric test................ Right............ Left ....
Rotation after nystagmus " .... " ....

REMARKS.

Urine

Blood

Diagnosis ______ Prognosis ______
Remarks.

Treatment.

Formulæ.—Appended below is a short list of formulæ, or prescriptions, which in the author's experience have proved their value in practical otology.

ALKALINE AND ANTISEPTIC SOLUTIONS FOR CLEANSING THE NASAL AND NASOPHARYNGEAL SPACES.

1. ℞ Sodii bicarbonat.,
Sodii borat.,
Sodii chlorid., āā gr. x;
Aquæ dest., oz. j.

Sig.—A non-irritating alkaline spray useful in cases in which all antiseptic drugs, even in weak solution, prove irritating.

2. Sodii bicarbonat.,
Sodii borat., āā gr. xl;
Glycerini, oz. j;
Aquæ dest., ad oz. iv.

Sig.—Mildly astringent alkaline spray useful in some cases of nasopharyngeal congestion.

3. ℞ (Dobell's solution).
Acid. carbolic., gr. iv;
Sodii bicarbonat.,
Sodii borat., āā gr. xl;
Aquæ dest., q.s. oz. iv.

Sig.—Antiseptic spray useful in bacterial infections of nose or nasopharynx.

4. ℞ Alkalol.

A proprietary preparation, alkaline, non-irritating, and slightly astringent; may be used as nasal spray undiluted.

OIL SOLUTIONS.

5. ℞ Menthol, gr. iij;
Albolini, oz. j.

Sig.—Nasal spray.

6. ℞ Menthol, gr. x;
Albolini, oz. j.

Sig.—Nasal spray.

7. ℞ Menthol,
Camphor, āā gr. vj;
Albolini, oz. j.

Sig.—Nasal spray.

N. B.—The use of an aqueous solution in the nose should be immediately followed by an alboline spray, the coating of oil thus supplied being distinctly protective to the mucous membrane, particularly if the patient is going immediately out into the open air.

The use of the weaker or stronger menthol solution depends in part upon whether it is intended to allay congestion or mildly to stimulate the mucosa; and also to some extent upon individual differences in susceptibility to the local action of the drug.

The addition of camphor is useful in some cases in relieving turbinal congestion.

Astringent and Antiseptic Solutions for Direct Application to the Pharyngeal Mouth of the Eustachian Tube.

8. ℞ Argenti nitratis, gr. x;
Aquæ dest., oz. j.

Sig.—Apply locally by means of cotton applicator

9. ℞ Argenti nitratis, gr. xx;
Aquæ dest., oz. j.

Sig.—Apply locally.

10. ℞ Argenti nitratis, gr. xxx;
Aquæ dest., oz. j.

Sig.—Apply locally.

N. B.—Nitrate of silver in the above strengths is well borne by the nasopharyngeal and tubal mucosa. It is best, however, to begin with the weakest solution (gr. x ad oz. j) and gradually increase the strength of the solution. A solution of 30 grains to the ounce has in the writer's experience been as strong as can be advantageously used in this region. In using the Yankauer or the ordinary wire applicator for applying nitrate of silver solutions throughout the entire length of the canal, a solution of 10 or 20 grains to the ounce is as strong as should be used.

11. ℞ Argyrol, 25 per cent. solution.

12. ℞ Argyrol, 50 per cent. solution.

13. ℞ Ichthyol, 50 per cent. solution.

N. B.—As substitutes for silver nitrate the above are very useful. Argyrol is strongly antiseptic and at the same time practically non-irritating. For application throughout the length of the Eustachian tube, argyrol in 25 per cent. or 50 per cent. solution is a safe drug which exerts a distinctly beneficial influence in many cases of tubal catarrh.

Ointments.

(Used in otological practice chiefly in the treatment of eczematous affections of the auditory meatus and auricle.)

14. ℞ Unguent. zinc. oxid., oz. ss;
Petrolati, q.s. ad oz. j.—M.

Sig.—Local application in acute auricular eczema. The parts should be kept constantly coated with the ointment.

15. ℞ Ichthyoli, dr. j;
Unguent. zinc. oxid., dr. iij;
Petrolati, q.s. ad oz. j.—M.

Sig.—Indicated in acute auricular eczema with denuded surface or considerable inflammatory reaction.

16. ℞ Acidi carbolic., gtt. iij;
Unguent. zinc. oxid., dr. iv;
Petrolati, q.s. ad oz. j.—M.

Sig.—Indicated in acute auricular eczema with excessive secretion of pus.

17 ℞ Unguent. hydrarg. oxid. rub., dr. ij;
Unguent. aquæ rosæ, q.s. ad oz. j.—M.

Sig.—Indicated in chronic eczema of meatus or auricle.

Cleansing Fluid for Irrigating the External Auditory Meatus.

18. ℞ Acid. carbolic., 1 in 200 solution.

Sig.—For irrigating meatus preparatory to myringotomy or tympanic operation; not suitable for routine use.

19. ℞ Hydrargyri bichlorid., 1 in 2000 solution.

Sig.—For cleansing the auditory canal preparatory to myringotomy. Bichloride of mercury even in weak solution is, in the author's opinion, too irritating for routine or home use.

20. ℞ Acid. boric., oz. iv.

Sig.—Dissolve half a drachm in cup of boiled water. A non-irritating solution which is the best irrigating fluid for routine cleansing of the ears.

Solutions for Instillation into the Ears.

21. ℞ Sodii bicarbonat.,
Sodii biborat., āā gr. x;
Aquæ, oz. j.

Sig.—Good solvent of hardened cerumen, when latter cannot otherwise be removed without injuring canal.

22. ℞ Acid. boric., gr. xx;
Alcohol, 95 per cent., oz. j.

Sig.—Fill auditory canal and retain 4 to 5 minutes. A cleansing solution which promotes healing in purulent otitis media after the acute stage has passed.

23. ℞ Acid. carbolic., gr. ij;
Acid. tannic., gr. x;
Aquæ dest., oz. j.

Sig.—Ten or twenty drops in ear night and morning. Useful in certain cases of subacute otitis media, with only moderate discharge.

24. ℞ Acid. salicylic., gr. x;
Alcohol, oz. j.

Sig.—Fill auditory canal night and morning and allow to remain 5 to 10 minutes. Almost a local specific against the various forms of aspergilli occasionally lodging in the ear.

Local Anæsthesia for Operation of Myringotomy.

25. ℞ Cocain. hydrochlor.,
Acid. carbolic., āā gr. xlviij;
Aquæ dest., oz. j.

Sig.—A small round pledget of sterile absorbent cotton is saturated with this fluid and the excess shaken off. It is then placed in contact with the drum membrane and allowed to remain about two minutes. Drum membrane should then be immediately incised. In some cases absolutely no pain is felt; in others some pain is experienced.

Probably in all cases the pain is lessened. While stronger solutions of carbolic acid have been advised and will more surely induce local anæsthesia, there is distinct danger of permanent injury of the drum membrane by carbolic solutions of greater than 10 per cent. strength. Even in this strength the solution is advised only in cases in which nitrous oxide cannot be given, general anæsthesia, in the writer's view, being always preferable.

For the Relief of Moderate Pain.

26. ℞ Codeinæ, gr. j;
Phenacetini,
Salol, āā gr. xx.

M. Divid. in capsul. No. viij.

Sig.—One capsule t. i. d.

Certain cases characterized by persistence of moderate ear pain even after incision of the drum-head are relieved by two or three doses of the above.

For the Relief of Nasopharyngeal and Tubal Congestion.

27. ℞ Extract. belladonnæ, gr. j;
Salol, gr. xx.

M. Divid. in capsul. No. viij.

Sig.—One capsule q. 4 h.

Rheumatic or plethoric patients suffering from acute suppurative otitis media often exhibit intractable nasopharyngeal congestion. In such cases this formula is often of value.

28. ℞ Tablets (Lincoln's formula) each to contain
Extract. belladonnæ, gr. 1/8;
Camphor., gr. 1/4;
Quinin. sulphat., gr. 1/8.

Sig.—One tablet q. 3 h. Useful in reducing obstinate nasopharyngeal congestion.

29. ℞ Extract. rhei fluid.,
Sodii bicarbonat.,
Spirit. piper. menth., āā dr. j;
Aquæ dest., q.s. ad oz. iv.

Sig.—One teaspoonful in wineglass of water t. i. d., a. c.

This well-known formula is repeated here chiefly on account of the author's experience as to its rather wide range of usefulness. For children with acute auricular eczema, plethoric adults suffering from furunculosis of the meatus, and rheumatic individuals suffering from subacute tympanic disease, there is no question that the administration of this mixture is often a positive factor in bringing about recovery.

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